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CONTENTS

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BRITISH JOURNAL OF TUBERCULOSIS AND DISEASES OF THE CHEST

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GENERAL ARTICLES

THE PSYCHOLOGICAL ASPECTS OF AFTER-CARE IN PULMONARY TUBERCULOSIS*

By Eric D. Wittkower

From the Allan Memorial Institute of Psychiatry, Montreal, Canada

I. The Personality of the Tuberculous Patient

A discussion of this subject may be appropriately preceded by a brief résumé of findings concerning the personality of tuberculous patients and changes effected by the illness and its treatment.

Investigations have failed to detect a uniform personality type prevailing in tuberculous patients, but they have shown that an inordinate need for affection is a common and outstanding feature of their premorbid personality. As one might expect, this not infrequently gives rise to conflicts over dependence and aggressiveness. Reconstruction of their personalities reveals that some of these individuals display their need for affection and allied dependent trends overtly—one may almost say in a blatant form—whereas others conceal them behind such psychological defences as self-assertiveness, ostentatious independence, excessive self-drive and aggressive or provocative behaviour.

Situations which entail a denial of love, a rebuff or a separation from, fear of losing, and actual loss of a much-needed supporting figure often precede the onset of tuberculosis and its relapses.

The *illness* itself gives rise to such regressive phenomena as secondary narcissism, egocentricity, hypochondriasis, over-dependence and demanding need for affection. Tuberculous patients have these phenomena in common with any other group of persons disabled by chronic illness or injury.

But there are some features typical of tuberculous patients. Because their hopes and aspirations have crashed they feel depressed and bemoan their losses; because tuberculosis is an insidious disease they are in an anxious mood varying in intensity; and because tuberculosis is an infectious disease they feel—and are to a certain extent—outcasts of society. Some patients camouflage their feelings of anxiety and of depression by a manic or defiant façade, while others project their resentment at their condition on to their

* Based on an address given at the Regional Conference on Chest Diseases of the American Psychiatric Association, at Montreal, Canada, March 7, 1952.

doctors, nurses, sanatoria or the world in general. Underlying anxiety in its turn leads to over-dependence. Frightened children and those in need of affection cling to their mother's apron strings.

Added to the effects of the illness are the effects of the *treatment*. Even if at a later stage surgical measures should be decided upon, tuberculous patients have to subject themselves for prolonged periods to dependence on doctors and nurses and to a life of isolation, of complete inactivity or at least of enforced idleness. Hence constriction of the field of interest, an inevitable result of the disease narcissism, is intensified by measures prevailing in the treatment of tuberculosis.

One of the tasks of after-care therefore seems to be to undo the psychological damage done to the patient by his prolonged illness and his hospitalisation. Factors which require special consideration are: dependent trends, feelings of inadequacy, of anxiety and of depression, and the sense of isolation.

II. Specific Problems of After-care

Let us now turn to the specific problems of after-care.

Every patient goes, of course, through a difficult period after discharge from the sanatorium, owing to the sudden change-over from a sheltered regulated life to one without supervision. Suddenly deprived of the protective tutelage of the sanatorium they tend to feel uncertain of themselves, insecure and anxious. If all goes well, this phase passes off sooner or later. But if the detachment from the sanatorium and its doctors has failed to materialise patients continue to feel foreign bodies in a healthy world.

In view of the long waiting lists nowadays, patients usually leave sanatoria incompletely recovered. If their recovery continues they have to re-adapt themselves—

(*a*) to being healthy;
(*b*) to life in a healthy environment; and
(*c*) to resumption of work.

(*a*) *Readaptation to Health*

Readaptation to health is a slow and protracted process. Years may have gone by and yet the one-time patient still views with misgivings a harmless rise of temperature, a slight coughing fit, a transient loss of appetite and a meaningless feeling of tiredness.

Chest physicians are familiar with patients who are unusually—and sometimes abnormally—concerned with the possibility of a relapse. These patients cling to their doctors on the rationale that any deterioration in their condition will be discovered at the earliest opportunity. Some of them are so much afraid of the possibility of a relapse—despite a " clean bill "—that they avoid the slightest exertion, sometimes in an absurd manner. More often than not these patients fail to respond to reassurance because their anxiety, although apparently concerned with and related to their illness, really arises from another source which requires exploring. Conversely, there are other patients who at an early date, defiantly and against their own interests, keep away from their doctors because they want to forget all about their illness.

(b) Readaptation to Healthy Environment

Apart from the factors mentioned, difficulties arise for ex-patients on return to a healthy environment—if it is a healthy environment—from their reduced wage-earning capacity, from their lowered physical strength and vitality, and from their re-entry into the family circle, the circle of previous friends and society in general.

Financial deterioration, an almost inevitable effect of the illness, is a frequent source of distress and of friction. It is easy enough to stick to one's husband if he is out all day and brings his wage packet home at the end of the week; but a husband who hangs about all day and does nothing and earns nothing may be more than many a woman can stand. If the breadwinner happens to be the sufferer, impairment of his prestige in the family structure can hardly be avoided. Pathetic is the situation of men whose social status has gone down as the result of their illness. Gradually the furniture becomes frayed, the paint flakes off the ceiling, the clothing becomes shabby, with precious little money left to effect any replacement. The ex-patient, understandably though unjustifiably, blames himself for this state of affairs and is apt to suspect accusations and recriminations where none exist.

Members of the family, if they are understanding, disregard but do not ignore the ex-patient's handicaps. Instances of families have been observed in which the homecomer has been subjected to manifest or, perhaps even more painfully, to concealed fear of infection. There are the mother and wife who continually bother—and irritate—the one-time patient by over-solicitude, as well as the inconsiderate husband or wife who leaves the semi-invalid and seeks his own amusements, or even accuses the convalescent of overdoing caution. In this way they " rub it in " that the homecomer is not—or at least not yet—at par with them. There are some marriages in which the past illness is never mentioned, but in which the unspoken fear of a relapse creates an artificial, tense and sometimes almost unbearable atmosphere.

Few convalescents enjoy leading a life of leisure. Usually they feel a burden to members of their family. Some complain bitterly about lack of consideration, while others allow themselves to be dragged into strenuous activities rather than be a deadweight on their healthy husband or wife or a spoilsport to their friends—not infrequently with disastrous results.

Particularly distressing for married women in their thirties is their doctor's advice not to have children for some time to come. They feel that their chances of ever having a child are rapidly passing, and that by being unable to have a child they are failing in their function as a woman and defaulting in their duty as a wife. Fear of impregnation not infrequently interferes with satisfactory consummation of sex relationships in these cases.

The world has, of course, not stood still while the patient was away at a sanatorium. Meanwhile husband or wife may have formed new ties, the healthy wife may have become self-reliant, the children may have grown up and may regard the homecomer as an intruder. Tuberculosis is a disease of the prime of youth. While the patients were away their contemporaries may have forged ahead, may have fallen in love and may have married. No wonder that, comparing herself with the more fortunate ones, many a young woman asks herself whether she will be left on the shelf. Loss of friends, if it

occurs, may be due to many reasons; tuberculous patients are apt to think it can be due only to one reason—the illness.

Society places a high premium on fitness. Anybody who is unfit, who is not at par in competitive value, potentially dangerous and, at any rate, different in his habits, as is the case with tuberculous ex-patients, exposes himself to suspicion if not contempt. Hence, looking fit but not being fit, many ex-patients are apt to strike a compromise between the doctor's advice to avoid exertion and their own desire to avoid awkward and sometimes painfully humiliating circumstances. Some succeed in making a social adaptation on their own terms, whereas others withdraw into a world of fellow sufferers.

(*c*) *Readaptation to Resumption of Work*

Persons who had previously been workshy can obviously not be expected to be first-rate workers simply because they have suffered from tuberculosis. They usually make the most of their disability and complain that excessive demands are made on them. Anxious individuals seek and content themselves with inferior jobs rather than endanger their precious health. A depressive mood, arising from thwarted ambitions, paralyses a person's initiative and, like resentment, prevents him from making a wholehearted effort. Persons who throw reasonable caution to the wind because they deceive themselves about their handicaps, or because overdefiantly they strive for more than they should do, not infrequently choose unsuitable occupations. Both attitudes may lead not only to disappointment but in some instances to a breakdown.

Adverse outside factors beyond the control of the disabled persons are: financial stress, erroneous vocational guidance, employment in unsuitable and especially inferior jobs, ostracism at work and bad management at their place of employment.

Particularly unfortunate are unskilled labourers. Some of them resume their previous—now unsuitable—occupation out of stupidity, out of necessity, or on account of guilt over having let their family down. Others, partly on their own initiative and partly on the advice of their doctors, set out in a search for a light open-air job, of which there are regrettably few, and land themselves in such occupations as gardener, lorry driver, window cleaner and park attendant. The suitability of some of these occupations on clinical grounds may be doubted; on psychological grounds the beneficial physical effect may be cancelled by the soul-destroying effect of monotonous employment, such as in the case of the man who was appointed superintendent of a cemetery which had not been in use for over sixty years.

Many firms are reluctant or refuse to employ persons with arrested tuberculosis, for a variety of reasons. Consequently ex-patients not infrequently find themselves in the predicament of either concealing their past history and obtaining an unsuitable job or revealing it and being rejected. Our experience has been that the breakdown-rate is much higher among those who conceal their past illness than among those who reveal it. Ostracism at work may be marked on resumption of work, but it loses its importance once the ex-patients have settled down. Of 120 employees who had returned to work for over three months only three related unpleasant experiences.

III. Practical Conclusions

Some practical conclusions may be permissible.

After-care, if it deserves to be spelt with a hyphen between the two words, should not be confined to the patient's physical condition. It should take into account his unwarranted feelings of anxiety, depressive features and dependent trends arising from the illness and from hospitalisation.

Feelings of anxiety, often of a phobic nature, may be precipitated but are not necessarily caused by the illness. Over-indulgence and over-protection on the part of doctors and nurses foster dependent trends which may have pre-existed the onset of the illness, whereas over-severity in patient management makes for defiant and even foolhardy behaviour.

Occupational therapy in its traditional form is of little avail in counteracting the demoralising and de-socialising effect of prolonged hospitalisation. Even for the most enthusiastic disciple toy-rabbit making loses its attractiveness after several years. There is a playful element in traditional occupational therapy. As the patient's stay at the sanatorium draws to its end, the more his activities approximate his future occupation the better suited they are to prepare him for the tasks which lie ahead of him.

A trial period at home some weeks prior to the final discharge from the sanatorium may help a great deal to lessen the inevitable anxiety resulting from a change-over from the world of the sick to the world of the healthy.

Prior to discharge a heart-to-heart talk of the medical superintendent or one of his senior medical officers with the patient and with members of his family seems essential. A discharge leaflet hardly seems adequate.

Vocational guidance of the tuberculous is still very much in its infancy. In the past it has been a common practice to give vocational guidance to patients mainly on the basis of their physical condition, and on the basis of what they wish to do. However, presence or absence of drive on the part of the patient, fear of doing too much or desire to do more than is wise, hysterical exploitation of and compulsive urge to disregard handicaps, are all factors which ought to be taken into account. In every walk of life there are plenty of self-selected unsuitables, but, in the case of the disabled, choice of the wrong job may be of serious consequence.

The view is generally accepted that tuberculous workers cannot be easily absorbed by normal industry. Home industries, though perhaps practicable in rural areas, are usually hamstrung and not really profitable through lack of capital. The solution of the problem seems to be sheltered employment. However, sheltered employment—hence its designation—entails continuation of protection in the work environment. Such protection may be necessary but should certainly not be perpetuated, for the reasons given, beyond the limits imposed by the patient's physical condition.

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METHODS OF RECORDING THE SHAPE OF THE CHEST

By H. R. E. Wallis

From the Department of Child Health, Bristol University

The shape of the human chest varies with age and occupation; and it may be affected by many diseases. Disorders of bone, cartilage, muscle, pleura, lung, heart or nerve may alter its contours. Therefore in many branches of medical research it is important to make records of this shape. In childhood the chest wall is at its most pliable, and such records are of great value in the study of child health and disease.

It is proposed to review the methods available for recording the shape of the chest, and to describe two new methods which may prove to be of value in the future.

For many years the form of the chest was determined by inspection and palpation alone. Description of the form was made in graphic terms having as their origin the pursuits of ornithology, cooperage and ecclesiastical ritual. For instance, chests were grouped into normal, those with features indicating proclivity to lung diseases (phthinoid, alar and flat chests), those with features indicating past diseases (rhachitic, pigeon breast and rosary, and Harrison's sulcus), and those with features indicating present disease (barrel-shaped and hollowed). Local changes were described as bulging, shrinking and funnel-shaped depressions. These harmless and somewhat medieval descriptions still appear in textbooks, and they still serve their turn as stumbling-blocks in qualifying examinations.

Mensuration

Measurement should be more accurate; but measurement of such an oddly shaped portion of the body, an irregular frustum which ceaselessly expands and contracts in several directions, is bound to be complicated. Measurements are usually made with a tape measure or callipers. These methods have been described by Myers (1927) and Roper (1948). The chest circumference is usually taken at the level of the fourth costal cartilage; but Parmenter and Gray (1922) showed that the circumference at the level of the tip of the xiphoid process is less subject to changes from obesity, mammary development and emaciation. These authors suggest that an average should be struck between the measurements in deep inspiration and complete expiration; but Dreyer (who has published much work on the vital capacity of the lungs) prefers one reading taken during quiet breathing. Myers (*loc. cit.*) points out that measurements of the expansion of the chest are not very reliable, as the amount of diaphragmatic action may vary, and a decrease on one side of the chest may be compensated for by an increase on the other. He suggests that a divided tape measure should be used—*i.e.*, one which reads from zero in the centre outwards in both directions. This enables one to read the difference in size of the two sides as well as the difference in the amount of expansion.

Callipers may be used to measure the diameters of the chest or the amount of respiratory expansion in the antero-posterior and lateral directions. But as the extent of these respiratory movements is small (less than 1 inch) in children, and the errors in reading (due to the callipers tilting, or the points pressing into soft tissues) are large, the measurements are not of great value. Another measurement which is sometimes made is that of the sternal angle. This is small in the phthinoid and large in the emphysematous type.

Cyrtometry

A cyrtometer is an instrument for measuring and recording curves. The cyrtometers used in medicine are usually made of strips of soft metal which are bent to fit the contours of the chest wall and then placed on paper so that a tracing may be made. Myers (*loc. cit.*) recommends the use of two strips of soft lead 2 feet long, $\frac{1}{4}$ inch thick and $\frac{1}{2}$ inch wide, hinged together with rubber tubing. In practice this is rather cumbersome, and it is convenient to use one strip only and to record each side of the chest separately. (The mid-points of the chest are marked with skin pencil.) This method has the advantage that tracings of the two sides of the chest may be more easily compared. Fig. 1 shows a tracing made with the lead strip cyrtometer from a child with a congenital funnel chest. The tracings may be made on squared paper, which enables accurate comparisons to be made (Fig. 9), or one side may be reversed and superimposed on the other, as in Fig. 10. Tracings may also be made of the vertical contours of depressions and protrusions (Fig. 11).

Disadvantages of this instrument are that the lead is rather stiff and needs to be moulded, especially when recording a depression. When making tracings of young children, whose ribs are very pliable, the ribs may bend before the lead does. The method is tedious and dirty. Lead dust comes off on to the hands of the operator, thus exposing him to the risk of a new occupational disease—Chest Physician's colic. Search was made for another metal, but it soon became apparent that two contradictory qualities were required—namely, pliability (which is needed when fitting the instrument to the chest) and rigidity (when transferring the shape to paper). Dr. N. Thompson, Lecturer in Physics at Bristol University, suggested the use of an adjustable template. This is an instrument used by masons for copying mouldings in stone. Accordingly an apparatus was constructed, and found to be of value.

The Multiple Rod Cyrtometer

This apparatus, which is shown in use in Fig. 2, consists of two solid oblong plates of ebonite 14 inches long and 3 inches wide. The lower plate is grooved transversely, and in the grooves lie thirteen dozen aluminium rods each of $\frac{1}{13}$ inch diameter and 9 inches long. (These rods may be obtained from the manufacturers of a well-known brand of child's building set.) On top of the rods is a strip of sorbo rubber, and then the upper plate. Above this are two pieces of metal which help to spread the pressure evenly, then a bar of metal which keeps the plates rigid. Two nuts and bolts connect the various pieces and allow the rods to be made loose or firm.

In use the nuts are slackened until the rods slide freely in their grooves. The rods are then pressed gently against the chest and their ends become

adapted to the contour of the body. The nuts are tightened and the instrument is placed on a piece of paper. The curve may then be traced with a pencil or ball-pointed pen. A record made by this method is shown in Fig. 3. The instrument is remarkably quick and easy to use, and does not seem to be frightening to children. It has also proved of value when cutting linoleum to fit against moulded corners.

Harrison's Grooves

Naish (1947, 1948) made a simple apparatus for measuring the depth of Harrison's grooves (Fig. 4). The bar AB is laid across the groove, and the sliding rule C is pressed into the groove. The depth of the groove is measured on the scale. The sliding rule is $\frac{5}{8}$ inch wide, and its width prevents it from reaching the nadir of some grooves or funnel-shaped depressions. However, this is an advantage, as the rule does not sink into the normal depressions between adjacent ribs. The instrument gives no information about the width or slope of the groove. To record these details a cyrtometer tracing is needed.

The Posture Recorder

This instrument was designed by the late Mr. R. E. Roper, M.A., M.Ed., as an aid to physical training in schools. It consists of a movable lens, a mirror and a ground-glass screen, contained in a box with a flap which acts as a screen against the light. The patient is placed against a light background (a window, a white wall or a sheet), and an image of him is thrown on to the ground glass as in a reflex camera. Tracing paper is then placed over the glass and a tracing is made in pencil (Fig. 5). Either the whole child or the chest alone may be portrayed. This apparatus is of value for following the effect of remedial exercises on the posture. The co-operation of children is easily obtained if they are shown the tracings. Difficulty is usually experienced with young children as they cannot keep still for long enough and tend to sway about. Also personal errors of draughtmanship may influence the result. Fig. 5 shows a normal child, and Fig. 6 one with typical deformity associated with long-standing asthma.

Casts

Casts of the chest or of a portion of it may be made. They are of great value in following the development over a period of time of abnormal shapes, such as funnel chests, or the bulges associated with congenital heart disease. They may be constructed from plaster of Paris, gauze and collodion, or some of the plastic substances used in making dental impressions. The disadvantages are that they take a long time to make, and the process is frightening to young children. They are expensive, and when finished are difficult to store.

Photography and Radiography

Photography is useful, but does not lend itself to accurate measurement. The results of attempting to demonstrate Harrison's grooves are sometimes unconvincing (Figs. 8 and 14). Funnel chests are more easily demonstrated. Radiography may be of value in showing localised deformities of the ribs or spine, and the inclination and spacing of the ribs in emphysema and collapse.

FIG. 2.—The Multiple Rod Cyrtometer in use.

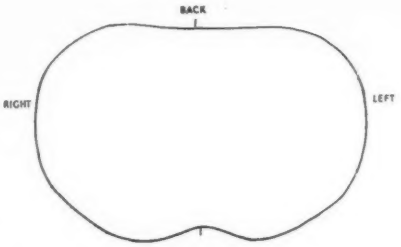

FIG. 1.—Tracing made with the lead strip cyrtometer, showing funnel chest. Case 3, boy aged 4 years. *See also* FIG. 16.

FIG. 3.—Tracing made with the Multiple Rod Cyrtometer, showing congenital deformity of the chest. Boy aged 4½ years.

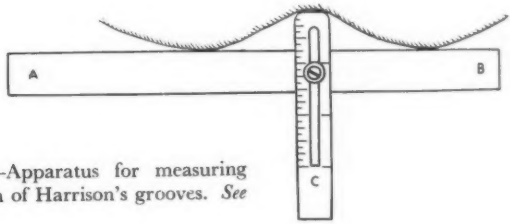

FIG. 4.—Apparatus for measuring the depth of Harrison's grooves. *See text.*

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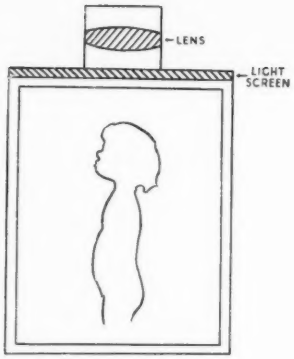

FIG. 5.—Plan view of the Posture Recorder with image.

FIG. 6.—Tracing made with the Posture Recorder. Boy aged 6½ years, chronic asthmatic.

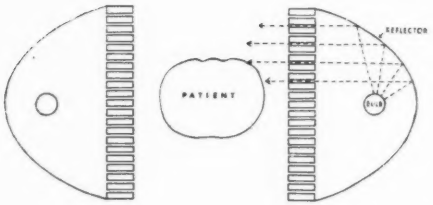

FIG. 7.—The Optical Contour Apparatus, horizontal section.

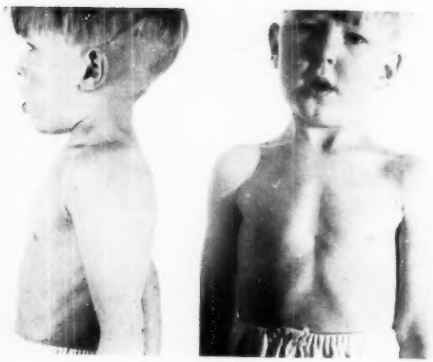

FIG. 8.—Case 1, boy aged 4½ years. Bronchiectasis of left lower lobe and lingula.

PLATE XIV

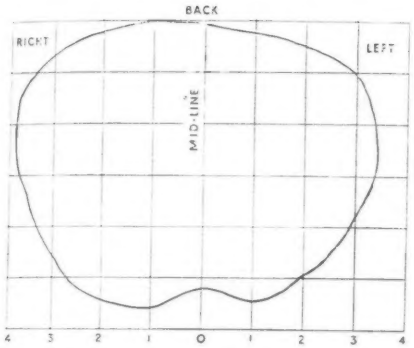

FIG. 9.—Tracing on squared paper of the chest of Case 1.

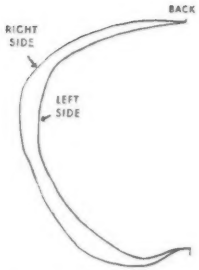

FIG. 10.—Case 1; the two halves of the chest superimposed.

FIG. 11.—Case 1; vertical tracing through Harrison's groove.

FIG. 12.—Case 1, under the Optical Contour Apparatus, showing Harrison's groove on the left.

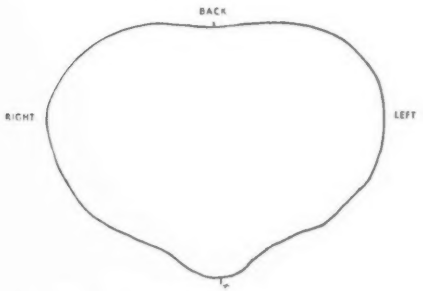

FIG. 13.—Tracing made with the lead strip cyrtometer of the chest of Case 2. Boy aged 5 years, asthmatic.

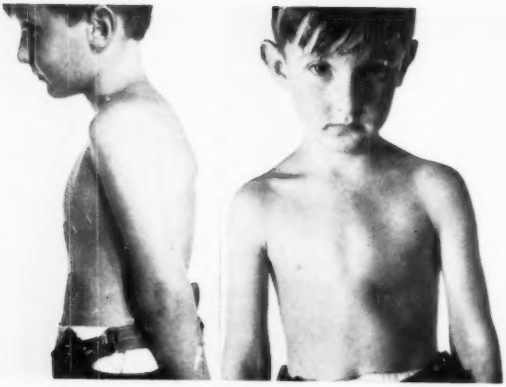

FIG. 14.—Case 2. Boy aged 5 years, asthmatic.

FIG. 15.—Case 2, under the Optical Contour Apparatus, showing bilateral Harrison's grooves, pigeon chest and sternal depression.

FIG. 16.—Case 3, under the Optical Contour Apparatus, showing funnel chest and Harrison's grooves.

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Lateral views show pigeon breasts and funnel chests; and stereoscopic radiography might be expected to demonstrate funnel chests also, though its use would be supererogatory.

The Optical Contour Apparatus

This apparatus was designed by B. N. Wallis, Esq., C.B.E., F.R.S., Chief of Research and Development at Vickers-Armstrongs Ltd., Weybridge. It was produced in order to facilitate the carving of portrait heads in wood. A cross-section of it is shown in Fig. 7. It consists of two lamps each containing two "Photoflood" bulbs and a parabolic reflector. The light from the bulbs and the reflector passes through a grid of plywood strips. There are forty-eight of these strips and forty-seven spaces between them, though to make the task of drawing easier a smaller number of strips has been portrayed in the illustration. The strips are approximately $\frac{1}{4}$ inch wide, and the spaces between them $\frac{1}{32}$ inch. The suspension of the lamp chambers is so arranged that opposed planes of light are parallel and coincident when they meet. The subject for study is placed between the grid boxes and is thus illuminated from both sides by vertical strips of light which are a set distance apart. These strips of light on the subject follow the contours of the body, and by this means depressions and projections are demonstrated.

Examples

(1) The child R. S., a boy aged $4\frac{1}{2}$ years, suffered from bronchiectasis of the left lower lobe and lingula. Clinical examination showed diminution in size and flattening of the left hemithorax, with a Harrison's groove on the left $\frac{5}{16}$ inch deep and a funnel chest $\frac{1}{4}$ inch deep. The circumference of the chest at the level of the xiphisternum was 21 inches in expiration and 22 inches in inspiration. The difference between the two sides was considerable—namely, right 11 inches, expansion $\frac{3}{4}$ inch; and, left 10 inches, expansion $\frac{1}{4}$ inch. Fig. 8 shows the standard photographs of this child. The funnel chest is evident, but of the other features there is scarcely a hint. Fig. 9 is a tracing of his chest at the level of the xiphisternum made with the lead strip cyrtometer and traced on to squared paper. Fig. 10 shows the two halves of the chest superimposed, and Fig. 11 the vertical contour of the Harrison's groove. In Fig. 12 the boy is seen under the optical contour apparatus. The groove can be seen very clearly, and also a certain amount of falling in of the chest wall below the left clavicle.

(2) T. F., a boy aged 5 years, suffered from asthma for two years. The shape of his chest is difficult to describe. It shows marked Harrison's grooves. The depth of these grooves is: right $\frac{5}{8}$ inch; left $\frac{3}{4}$ inch. There is also a pigeon chest, shown in Fig. 13 (a tracing at the level of the xiphisternum), while higher up the sternum there is a depression. Fig. 14, standard photographs, shows only the groove on the left and the central depression. Fig. 15, taken under the optical contour apparatus, shows both grooves, and it is evident that the one on the left is deeper than that on the right. The dark central area is caused by the depression in the sternum, and the V-shaped contour of the lines below it indicates the pigeon chest.

(3) Fig. 16 is an oblique view of a child with a severe congenital funnel

chest and bilateral Harrison's grooves. His father and grandmother had similar deformities.

Calculation

The optical contour apparatus may be used for very accurate recording of the shape of the chest and its movement with respiration. In the first place, a plane of light may be marked by occlusion of part of it with plasticine or adhesive tape. The distance between successive planes of light is known (it is 0·2826 inch). By counting the number of strips of light between a point on the chest wall and the marked plane the distance of the point in front of or behind that plane can be calculated. Simple trigonometry enables the lateral and vertical co-ordinates to be calculated.

Photographic Details

Two " Photoflood " bulbs are used in each lamp. O ving to the occlusion by the grid of a large part of the light the intensity of illumination on the subject is not very bright. It is hoped to overcome this in the future by the use of electronic flash bulbs.

Summary

A review has been made of the methods available for recording the shape of the chest. The following methods have been considered: verbal description, mensuration, cyrtometry, the posture recorder, photography and radiography, and casts. A new method of recording curves by the multiple-rod cyrtometer is described. The optical contour apparatus, which assists in the visual assessment of deformities, is also described, and examples are given of its use. The apparatus should prove of value in recording the movements of individual ribs in health and disease.

I wish to thank the following persons who have rendered this investigation possible: Professor A. V. Neale for his continued encouragement and advice; Dr. Beryl Corner and Dr. J. Apley for permission to examine children under their care at the Bristol Children's Hospital and the Royal United Hospital, Bath; Mrs. Roper for information about the posture recorder; Mr. B. N. Wallis for the loan of the optical contour apparatus, and for advice on technical details; Dr. J. M. Naish for the loan of his apparatus for measuring the depth of Harrison's grooves; Dr. Marcia Hall for information about plaster casts; Dr. Mary M. M. Boyd for assistance with collodion casts; Dr. N. Thompson for advice on the adjustable template; Mr. D. Allen for constructing the multiple rod cyrtometer; and Mr. Godman and the staff of Bristol University Photographic Department for advice and technical assistance.

The work was carried out during my tenure of the appointment of Assistant Medical Officer in the University of Bristol Health Service.

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THE TREATMENT OF PERSISTENT TUBERCULOUS PLEUROPULMONARY FISTULÆ

A REPORT OF FIVE CASES

By J. R. Belcher

From the London Chest Hospital

Since the introduction of chemotherapy and the more enlightened selection of cases for artificial pneumothorax in the treatment of pulmonary tuberculosis, the incidence of tuberculous empyema has fallen considerably, but it remains as an occasional serious complication in the course of the disease.

It is suggested that an empyema is frequently due to rupture or leakage of a tuberculous cavity into the pleura, and that in fact this accident may be the commonest cause of the complication. Auerbach (1949) goes so far as to say that this is the sole cause of tuberculous empyemata.

The records of the London Chest Hospital show that of the thirty-four patients treated for tuberculous empyema during the last six years, nineteen gave a history of onset very similar to those shortly to be described, and that only in the remaining fifteen did the complication appear with the development of frank pus in a pre-existing effusion without obvious fistula formation.

There are four ways in which pleuropulmonary fistulæ may develop in tuberculous patients:

(1) A cavity may rupture or leak spontaneously into a previously established artificial pneumothorax.
(2) A cavity may rupture or leak following damage to the lung at adhesion section or during a refill.
(3) A cavity may rupture or leak spontaneously before an A.P. has been established.
(4) An empyema may rupture into the lung.

An example of three of these has been encountered in the five cases described here. In all patients in whom a pleuropulmonary fistula persists it is difficult to obliterate the pleural space by aspiration (Siddons, 1951), the prognosis in untreated cases is bad, and even when treated late by surgery the morbidity and mortality is high (seven of the nineteen cases already mentioned died as a direct result of the accident). If it is recognised that this complication is by no means uncommon, particularly after adhesion section, and if it is diagnosed and treated early by resection, it is submitted that the high mortality and morbidity may be considerably reduced. Five patients who have been dealt with in this way are described below.

Case 1

M. C., a girl aged 24, first developed left-sided pleurisy in 1946; after this she was well until February 1949, when a second attack developed. This was accompanied by pain and fever. She also complained of recent hæmoptysis,

productive cough and loss of half a stone in weight. In the absence of a positive sputum a diagnosis of pulmonary cyst in the left lower lobe was made (Fig. 1), and she was admitted to the London Chest Hospital on 7.3.49.

Shortly after admission she developed more pain in the left chest. Her sputum was found to contain acid-fast bacilli. Bronchoscopy showed no abnormality. The fever gradually settled, but on 18.3.49 she was seized with sudden severe pain and tightness in the left chest and became very dyspnœic.

On examination at this time she was found to have a hydropneumothorax. The mean intrapleural pressure was +10, but after the removal of 300 c.c. of air her symptoms were considerably relieved. A diagnosis of acute rupture of a tuberculous cavity was made. One day later turbid, blood-stained fluid was aspirated and the radiograph showed a hydropneumothorax with the lung almost completely collapsed (Fig. 2). The fluid contained no secondary organisms and on culture showed no acid-fast bacilli. Repeated aspiration of air and fluid enabled the lung partially to re-expand, but an intermittent bronchopleural leak persisted. It was therefore decided that a left lower lobectomy should be undertaken. A course of streptomycin and penicillin had been started at the outset, and on 29.3.49—that is, ten days after the rupture of the cavity—a left lower lobectomy was performed. The pleura contained thick pus and there was a ¼-inch layer of fibrin covering it. The bronchopleural fistula was patent throughout the operation. The fissure was easily found and the lobectomy was not unduly difficult. No decortication of the upper lobe was undertaken.

After the operation chemotherapy was continued for twenty-eight days and a pneumoperitoneum was induced (the phrenic nerve was not crushed) and maintained for two months. The upper lobe expanded slowly, but eventually completely filled the pleural space. An X-ray taken 6.5.49 (Fig. 3) shows almost no evidence of pleural thickening. Despite the layers of infected fibrin left behind in the pleura after her operation, no fluid aspirated subsequently contained tubercle on culture.

She was last seen in December 1950, when she was well, at work, her sputum was negative and she was symptom-free.

Comment: Lobectomy was in this case undertaken early—that is, on the tenth day after the rupture of the cavity. No decortication of the upper lobe was done, but nevertheless expansion was complete and fluid aspirated after operation never contained acid-fast bacilli.

Case 2

N. B., a girl of 16, was first admitted to hospital in April 1949 with a diagnosis of miliary tuberculosis. This was treated with streptomycin and the disease largely cleared, leaving a right apical lower lobe cavity. For this an A.P. was induced in August 1949 (Fig. 4). No thoracoscopy was done, but the A.P. was maintained until 8.4.50, when she awoke complaining of severe pain in the right chest, and dyspnœa. She was seen five days later at her clinic, where she was found to be febrile, and where the intrapleural pressure, which was −10 +5, could not be altered by aspiration of large quantities of air.

She was admitted to Highgate Hospital with a temperature of 101° and a pyopneumothorax. Her sputum was minimal and negative, but a radiograph showed complete collapse of the lung with some effusion in the pleura (Fig. 5). 500 c.c. of straw-coloured fluid were aspirated. This contained a pure culture of pneumococci. She was treated with P.A.S. 18 grammes, streptomycin 1 gramme, and penicillin 1,000,000 units daily, and the pleura was aspirated

Fig. 1.—Case 1. Before cavity rupture.

Fig. 2.—Case 1. Immediately after rupture, showing hydropneumothorax.

Fig. 3.—Case 1. 9 months after lower lobectomy.

Fig. 4.—Case 2. With right apical lower lobe cavity and artificial pneumothorax.

Fig. 5.—Case 2. Showing pyopneumothorax after acute cavity rupture.

Fig. 6.—Case 2. After lobectomy and decortication, showing persistent pleural thickening.

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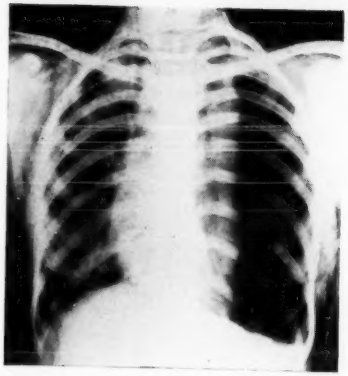

Fig. 7.—Case 3. 24 hours after adhesion section, showing extensive subcutaneous emphysema and a tension pneumothorax.

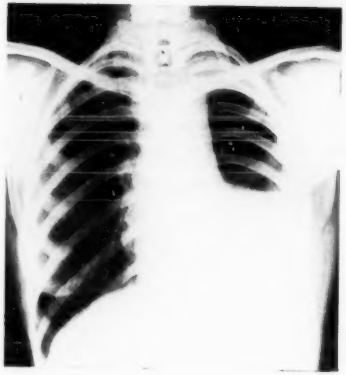

Fig. 8.—Case 3. 4 months later, showing total pyopneumothorax.

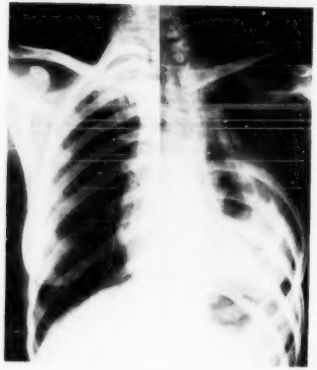

Fig. 9.—Case 3. 3 months after upper lobectomy, showing some re-expansion of the lower lobe and evidence of a broncho-pleural fistula.

PLATE XVII

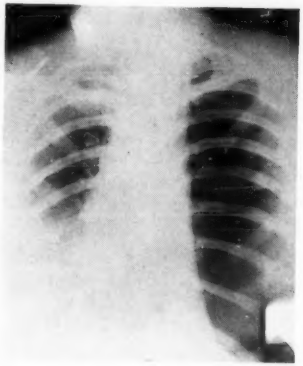

Fig. 10.—Case 4. Showing tension pneumothorax due to subacute leakage one month after

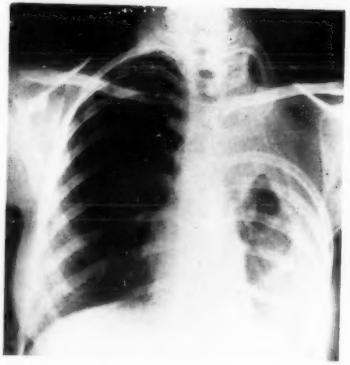

Fig. 11.—Case 4. 4 months after upper lobectomy and thoracolysis.

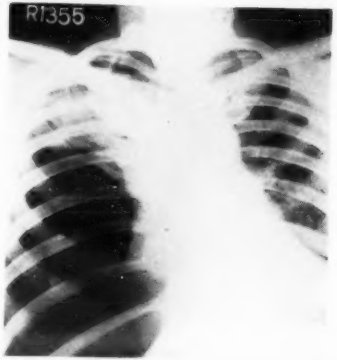

Fig. 12.—Case 5. Showing tension pyopneumothorax after acute cavity rupture.

every other day, penicillin being instilled on each occasion. The fever persisted, but the pneumococci disappeared from the fluid immediately. The effusion rapidly thickened and became thick green pus containing scanty acid-fast bacilli. A large fistula was not now present, as a negative pressure could be maintained after aspiration for up to twenty-four hours, but she had an intermittent pleuropulmonary leak. It was therefore decided, on 5.5.50, that she had had a rupture of a tuberculous cavity and that a small leak had persisted. Lobectomy and decortication were considered to be the treatment of choice. She was transferred to the London Chest Hospital on 7.5.50.

A right middle and lower lobectomy was carried out four weeks after the accident. The lung was completely collapsed, the pleura was covered with a thick fibrinous layer, and several ounces of thick pus were present in the space. There was an obvious leak of air from the apex of the lower lobe throughout the operation. The whole lung was decorticated without difficulty and the lobectomy was completed. The fibrinous layer on the parietal pleura was not removed.

On the next day the upper lobe was expanding well, but subsequently became atelectatic and a bronchoscopy had to be performed. The pleura was aspirated on several occasions, but never contained acid-fast bacilli, despite the fact that the fibrinous layer showed giant-cell systems on section. A pneumoperitoneum was induced on the fourth post-operative day, but was soon abandoned as it caused acute discomfort. P.A.S. and streptomycin were given for five weeks. Convalescence was very slow, but the lung re-expanded gradually and the remains of the air were absorbed.

When last seen, in September 1951, the patient was well, symptom-free, gaining weight and her sputum was negative. An attempt at aspiration of the pleura failed to reveal any pus, but the radiograph still shows that there is a considerable layer of thickened pleura covering the upper lobe (Fig. 6).

Comment: In this case the interval before operation was longer (one month). Decortication was easy, but expansion was slower than in the previous case and not so complete. Again, despite the presence of frank pus in the pleura at operation and infection of the pleura left behind, fluid aspirated after operation contained no acid-fast bacilli.

Case 3

D. R., a girl aged 23, was first found to have pulmonary tuberculosis in December 1948. She rested at home until her admission to Pinewood Sanatorium in January 1950. Her sputum was positive and her X-ray showed bilateral apical cavitation. A left A.P. was induced; three months later the adhesions were partially divided, and a second thoracoscopy was performed on 2.6.50. Two days later the patient was found to have widespread subcutaneous emphysema (Fig. 7). The lung was completely collapsed and stayed in that state despite repeated aspiration of large quantities of air. She had no pain or fever at this time, and fluid first aspirated seven days later was only slightly turbid, but acid-fast bacilli were eventually cultured from it after six weeks. It was thought that a persistent bronchopleural communication must be present (Fig. 8).

Another thoracoscopy was performed in the hope that further division of adhesions might allow the fistula to close. No further division of adhesions was found to be possible and a phrenic crush was performed.

Fluid was aspirated every three or four days and 1 gramme of streptomycin was injected on each occasion. The effusion only became definitely purulent

Fig. 12.—Case 5. Showing tension pyopneumothorax after acute cavity rupture.

Fig. 11.—Case 4. 4 months after upper lobectomy and thoracolysis.

sion pneumothorax due to sub-acute leakage one month after

on 13.10.50—that is, four months later. Fever developed on the fourteenth day, but subsided quickly, only to appear again three weeks later, when it took three weeks to subside. After this the patient remained afebrile.

As the fistula persisted and frank pus was being aspirated, it was decided that an effort must be made to preserve function of her left lower lobe, particularly in view of the cavitation at the right apex, and that a left upper lobectomy with decortication of the lower lobe should be carried out. This was done on 2.11.50. The lower lobe was decorticated at that time without difficulty, and expanded well on inflation; the parietal pleura was not removed. The fistula in the upper lobe was obvious throughout the operation, and the pleural cavity contained a fair quantity of thick pus.

After operation a pneumoperitoneum was induced, but on the fourth day she developed a hæmoptysis and the lower lobe collapsed, due almost certainly to a bronchopleural fistula rather than to a leak from the lower lobe. This collapse persisted, although the hæmoptysis stopped, and on 7.12.50 thoracoplasty was undertaken. Segments of the second, third, fourth and fifth ribs were removed. The pleura was opened along the bed of the sixth rib, a large amount of clot was turned out and the lower lobe was again decorticated. No fistula could be demonstrated at this time. Drainage was discontinued after forty-eight hours. The left lower lobe gradually expanded to fill the remainder of the pleural cavity and good air entry was audible over it (Fig. 9).

Recently this patient had a further hæmoptysis. Her general condition slowly deteriorated and her fistula reopened. The apical cavity on the other side increased in size and it was necessary to drain the infected space on the left side. She died in December 1951.

Comment: This is an example of cavity LEAKAGE after adhesion section. It was treated late by lobectomy and decortication and was complicated by a fistula.

CASE 4

B. R., a girl aged 19, first developed a productive cough in December 1949. This was treated conservatively until October 1950, when she developed a hæmoptysis. At this stage she had bilateral upper lobe cavitation which was more extensive on the left side.

She was admitted to Highgate Hospital, where a course of streptomycin and P.A.S. was started. She improved considerably, her right side cleared completely, but an A.P. was induced on the left as the cavity persisted. Extensive adhesions were divided one week later and a further adhesion section was undertaken on 23.1.51. She was then transferred to Pinewood Hospital, having had one refill after this thoracoscopy.

On 26.2.51 the left lung collapsed completely and, despite prolonged efforts with daily aspiration and finally with continuous suction, it was found impossible to get the lung completely to expand, as an intermittent pleuropulmonary fistula was present (Fig. 10).

On 31.3.51—that is, one month after the lung collapsed—she developed a temperature and her general condition gradually began to deteriorate; fluid now appeared in the left pleural space. Low-grade fever persisted. Repeated aspiration showed a slightly turbid effusion, from which it was never possible to grow acid-fast bacilli. It was decided that she had a persistent subacute pleuropulmonary leak, and that therefore lobectomy should be undertaken.

19.4.51. At thoracotomy she was found to have a dense white layer of fibrin all over a collapsed upper lobe and part of the lower lobe. A small

pleuropulmonary fistula was present. Left upper lobectomy and decortication of the lower lobe was undertaken. Convalescence was smooth. A pneumoperitoneum was induced four days after operation, and one specimen of fluid aspirated from her chest at that time showed acid-fast bacilli. On 17.5.51 thoracolysis was performed, the second, third and fourth ribs being removed.

Several subsequent aspirations showed no acid-fast bacilli on culture, although sections of both parietal and visceral pleura removed at operation showed giant-cell systems.

Since operation her condition has improved, her sputum is negative. The left lower lobe is now filling the hemithorax, and no further fluid can be obtained at aspiration (Fig. 11).

Comment: This was a case of cavity LEAKAGE developing one month after adhesion section, treated successfully by early resection and decortication.

CASE 5

J. W., aged 42. He had been a diabetic for six years. Pain developed suddenly in the right side of his chest, associated with tightness and dyspnœa. The pain persisted and was associated with night sweats. He was ill at home for one month, during which time he lost 2 stones in weight and developed increasing cough and sputum without hæmoptyses.

Prior to this acute onset he had had vague cough and lassitude for several months.

On admission to Highgate Hospital he was wasted and very ill, fever varied between 100° and 102°, and he coughed up 2 oz. of positive sputum daily. Radiologically he had a hydro-pneumothorax with multiple apical adhesions and upper lobe infiltration (Fig. 12). His chest was aspirated on 10.2.51—initial pressure +4 +8. 6,000 c.c. of air were removed, leaving the pressure −10 −5, but five minutes later it had returned to −4 −1, and it was impossible to maintain a negative pressure in his right pleura from that day onwards. Pus aspirated at the same time contained 90 per cent. polymorphs, but had no acid-fast bacilli or other organisms in it. One week later, however, on aspiration, acid-fast bacilli were found. He was treated with penicillin, streptomycin and P.A.S. and local streptomycin every other day. His temperature settled to 99°-98° and his general condition improved a little.

He was transferred to the London Chest Hospital on February 19, 1951, with a diagnosis of acute ruptured tuberculous cavity with a persistent pleuropulmonary fistula.

On admission his sputum was positive, he was febrile and his general condition was poor. His chest was aspirated, a large quantity of air was removed and a patent bronchopleural fistula was demonstrated. The aspirated fluid was thin pus which contained tubercle bacilli. Bronchoscopy showed some reddening and œdema of the right main bronchus, but despite this it was decided to proceed with thoracotomy forthwith.

At operation on 26.2.51, immediately on opening the pleura a wide-open fistula was apparent. It was found in the lower edge of the anterior segment of the upper lobe. There was a thin layer of fibrin covering both upper and lower lobes and there were multiple thick upper lobe adhesions. At first it was thought that the disease was confined to the upper lobe and lobectomy was performed, but on further examination of the lower lobe extensive disease was detected, particularly in the apex. It was therefore decided to perform a pneumonectomy and the parietal pleura was not removed. A pneumoperitoneum was induced on the eighth day after operation. Sputum was by then negative.

On 19.3.51 thoracoplasty was performed; the first, second, third, and fourth ribs were removed and apicolysis undertaken. Seven days later definite signs of a right bronchopleural fistula developed.

The pleural fluid aspirated on 13.3.51—that is, before there was evidence of fistula formation—was sterile and contained no tubercle bacilli.

Second-stage thoracoplasty, with removal of fourth to eighth ribs, was undertaken. The pleura was opened and the presence of a fistula was confirmed. Subsequently the empyema was drained by resection of the eleventh rib and thoracoplasty was carried down to this level.

Since then he has gradually improved. The dosage of insulin has diminished. His sputum is negative and his fistula closed. Drainage continues but is lessening. Fluid aspirated post-operatively on 3.3.51 was, however, positive on culture, and guinea-pig inoculation test of specimens of pleural pus and of visceral and parietal pleura were all positive. The pus draining from the tube has shown mixed organisms since operation, but never any tubercle bacilli, and his sputum has been consistently negative.

Macroscopic specimen of the lung confirmed the operative findings.

Comment: This was a case of rupture of a tuberculous cavity treated by early resection, but complicated by a fistula.

Pathology

Discussion

The rupture or leakage of tuberculous cavities has been variously ascribed to rupture of adhesions, ulceration of the cavity following over-distension, and necrosis of the wall due to the damage to the blood supply sustained during adhesion section.

TABLE I

	Rupture	*Leakage*
Persistence of fistula ..	Early pyopneumothorax	Eventual pyopneumothorax
Closure of fistula ..	Empyema	"Spontaneous pneumothorax"
Lesion	Large communication with massive infection	Small leak with minimal contamination
Onset	Acute, with fever, and malaise with tension pneumothorax	Symptoms of tension pneumothorax only
Progress	Rapid deterioration	Slow deterioration after weeks or months
Infection with A.F.B.	Early and heavy	Late and scanty
Secondary infection ..	Uncommon	Rare
Pus formation	Early	Late

The accident is most common during the course of artificial pneumothorax therapy. In his series Auerbach found that 81 per cent. of tuberculous empyemata developed in this period, and in the combined series described here there were 29 out of 39 which developed during A.P. treatment. When rupture or leakage occurs in the absence of an A.P., as in Cases 1 and 5, it is presumably due to ulceration of a cavity before the pleura has become firmly adherent over it.

The commonest single cause of the complication is adhesion section. This may lead to cavity *rupture*, as in a case described by Sarot (1949), or more commonly to *leakage*, as in Cases 2 and 3 in this series. The accident may be due to the division of the very extensive adhesions and with them the blood supply to the cavity which may have been derived from the parietal pleura,

but it is more likely that the lesion follows actual damage to the lung by the cautery at the time of adhesion section, since symptoms often appear directly after the operation, as in Case 2 and Sarot's patient. Where a cavity has ruptured, massive infection of the pleura with acid-fast bacilli is likely, although none were found in Case 1. Immediate secondary infection is uncommon, as tuberculous cavities seldom contain organisms other than acid-fast bacilli, although it did occur in Case 2.

When cavity leakage has occurred there is no massive escape of acid-fast bacilli and it may in fact be a long time before they are isolated. Nevertheless, tuberculous infection develops in both parietal and visceral pleura and was demonstrable in section of both layers in Cases 3 and 4, and in the visceral layer in Case 1, in all of whom definite giant-cell systems could be found. This infection was more obvious when the "spill" had been massive, as in Case 5, in whom it was possible to culture bacilli from both layers of pleura removed at operation.

Clinical Presentation

A persistent tuberculous pleuropulmonary fistula may present in two separate clinical forms. Although graduation between the extremes occurs the varieties are sufficiently different to be divided into acute and subacute types. The difference in the clinical picture depends on the size of the leak, the degree of infection with tubercle bacilli and whether or not a tension pneumothorax or secondary infection develops.

TABLE II

	Type	*Operation*	*Delay*	*Comments*	*Result*
M. C. ..	Acute rupture	Lower lobectomy	10 days	—	Excellent
N. B. ..	Acute rupture	Middle and lower lobectomy	4 weeks	Slow re-expansion	Good
D. R. ..	Subacute leakage	Upper lobectomy	5 months	Fistula	Poor
B. R. ..	Subacute leakage	Upper lobectomy	8 weeks	—	Excellent
J. W. ..	Acute rupture	Pleuro-pneumonectomy	7 weeks	Fistula	Fair

The *rupture* of a tuberculous cavity leads to a large leakage, gross soiling of the pleura with acid-fast bacilli, and possibly to secondary infection. A tension pneumothorax may also develop. This accident is a distinct clinical entity, and as it may call for urgent treatment it is essential that the diagnosis should be made as soon as possible. The onset is sudden, with pain, high fever, malaise, tachycardia, dyspnœa and a feeling of tightness in the chest, to be expected from massive pleural infection coupled with pulmonary collapse and a tension pneumothorax. Where the accident follows an adhesion section extensive subcutaneous emphysema may also be observed. The acute rupture of a cavity is not necessarily followed by the persistence of the fistula.

When the pleuropulmonary fistula does persist, as in Cases 1, 2 and 5, the patient may go downhill rapidly, and it is therefore essential that frequent readings of intrapleural pressure should be taken so that the persistence of the fistula can be recognised early. Treatment can then be undertaken and

the differential diagnosis from a tuberculous empyema, which may be treatable by aspiration, can be made.

Leakage of a tuberculous cavity into the pleura (Cases 3 and 4), like the acute type, may occur either when an A.P. has already been established or spontaneously. The fistula may seal off soon after it has occurred and no infection of the pleura may develop; in this way the lesion may behave exactly as does a " spontaneous " pneumothorax in tuberculous patients. On the other hand, the fistula may persist, leading to a chronic pleuropulmonary fistula which eventually will give rise to a tuberculous pyopneumothorax. The essential feature in the differential diagnosis between " cavity leakage " and " spontaneous pneumothorax " is the demonstration of persistence of these pleuropulmonary fistulæ.

The onset is different from that of acute cavity rupture. The symptoms and signs are largely due to alteration of pleural mechanics rather than to infection, and fever and malaise may be slight. Secondary infection is uncommon, the findings of acid-fast bacilli may be late and the organisms scanty. Clinically a spontaneous pneumothorax occurs; owing to the action of a check valve mechanism the intrapleural pressure may exceed the atmospheric and a tension pneumothorax may develop, with considerable dyspnœa, shock and a feeling of tightness in the chest. Again, where this occurs after adhesion section, widespread subcutaneous emphysema may appear, as in Case 3. A pleural effusion soon develops, but it may be a very long time before it becomes frankly purulent. The majority of spontaneous pneumothoraces cease to leak after one or two aspirations of fluid and air and seldom require aspiration after ten days from the onset, although persistent leakage may be demonstrable for this period. If this leakage continues after fourteen days there is an increasing likelihood that it will become chronic and then empyema will be inevitable, although in Case 4 it was some weeks before the diagnosis of cavity leakage could be made with certainty.

Treatment

Once the diagnosis of a persistent tuberculous pleuropulmonary fistula has been made, whether this is due to cavity rupture or to leakage, early resection is recommended. This is particularly true in patients who would, in any case, have been considered suitable for resection.

There are several reasons for this view; first, it is very unlikely that the fistula will close without surgical intervention, and the prognosis in patients with tuberculous pyopneumothorax is notoriously bad. Secondly, the thoracoplasty which may be the alternative operation is often more extensive than strictly necessary for the pulmonary lesion, without at the same time giving any certainty that the empyema can be closed. Resection and decortication, which gives a shorter period of morbidity and may preserve more pulmonary function, is therefore the procedure of choice. Lastly, it is certain that once a persistent leak has been established nothing is gained by waiting to initiate treatment. A great deal of ground may be lost by the patient in a short time, rendering operation more hazardous. The cases in this series tend to show that the earlier the resection is undertaken the better the late result.

The principles which govern the extent of the resection are those which

apply where no fistula is present, but clearly the area of lung from which the leak originated must be removed. In Cases 1 and 2 lobectomy was ideal, as both would almost certainly have come to resection. In those which followed a thoracoscopy (3 and 4) the site of the leak was known, and the upper lobectomy which was carried out might well have been thought suitable even in the absence of a fistula. In Case 5, despite the fact that the site of the lesion was quite unknown, thoracotomy was thought to be clearly indicated, and it is urged that this procedure should be repeated in similar patients, the extent of the resection required being decided when the chest has been opened.

TABLE III

	Pre-operative	*Decortication*	*1st Week*	*2-4 Weeks*	*Later*
1 M. C. ..	Negative	None	Negative	Negative	Negative
2 N. B. ..	Heavily positive	Visceral	Negative	Negative	Negative
3 D. R. ..	Positive	Visceral	Positive	Negative	Positive
4 B. R. ..	Negative	Visceral	Positive	Negative	Negative
5 J. W. ..	Heavily positive	Partial parietal	Positive	Positive	Negative

The risk of persistence of the empyema would at first sight appear to be considerable, particularly after pneumonectomy, but from experience in these cases and in others where pneumonectomy rather than pleuropneumonectomy has been performed for tuberculous pyopneumothorax it seems that once the source of infection (the fistula) has been removed the pleura, assisted by local and general chemotherapy, is capable of dealing with any remaining organisms. In the case of 3 and 5 it is felt that the bronchopleural fistulæ were responsible for the persistence of the empyema rather than the original infection of the pleura. As will be seen, the pleural fluid when cultured or smeared showed rapid clearing of organisms in all save those with the fistulæ. Nevertheless, sections of the parietal pleura suggest that this should be removed at the same time as the diseased lung as a precautionary measure, although this is not absolutely essential. The removal of the visceral pleura of the residual lobe should be as complete as possible in order to encourage rapid re-expansion.

Summary

(1) Tuberculous empyemata are frequently due to cavity rupture or to cavity leakage.
(2) Five patients in whom this accident had occurred are presented.
(3) All were treated by resection with or without decortication.
(4) It is suggested that this is the procedure of choice and that the earlier the accident is recognised and treated, the better the result is likely to be.

My thanks are due to Mr. Holmes Sellors, under whose care they were admitted, for permission to publish three of these cases.

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DISPLACEMENT OF THE UPPER ŒSOPHAGUS IN PULMONARY TUBERCULOSIS

By C. P. Silver

From Papworth Hospital, Cambridge

Accounts of the radiology of the œsophagus are mainly concerned with intrinsic diseases of the œsophagus, its displacement in cardiac disease, and displacements due to enlargement of the mediastinal lymph nodes. There are few descriptions of the œsophagus in pulmonary tuberculosis, and tuberculosis of the œsophagus itself is a rare condition. Lockard (1913) was able to find 69 cases in the literature which could, with reasonable certainty, be classed as œsophageal tuberculosis. All were secondary to advanced, usually pulmonary, tuberculosis. Shanks and Kerley (1951) state that tuberculous ulceration of the mucosa is not uncommon in the terminal stages of advanced pulmonary tuberculosis and may be suggested by spasm with obliteration or alteration of the normal mucosal pattern. Diverticula produced by traction on the œsophageal wall by some contracting inflammatory focus are usually ascribed to tuberculous lymph nodes. These too are rare.

Deviations of the œsophagus in chronic pulmonary tuberculosis have rarely been referred to by British authors; indeed, the indices of the two largest and most recent books on tuberculosis do not mention the œsophagus. William Evans (1936) illustrated a deviated upper œsophagus due to fibrosis of the right upper lobe, the mediastinum appearing central. Tanturri and Lucioni (1934) investigated the œsophagus by barium swallow examination in 21 cases of pulmonary tuberculosis in all stages of the disease. They often found deviations affecting the upper œsophagus, or, less commonly, the lower. But these were not related to tracheal deviations. Sometimes widening or deformity of the œsophagus was observed. Tanturri and Lucione (1934) ascribed the œsophageal deviation in pleuro-pulmonary tuberculosis to the fibrosing process, and found the clinical signs almost non-existent. These authors did not refer to the appearance of the œsophagus in plain chest radiographs without a barium swallow.

The absence of symptoms in œsophageal deviation is responsible for the lack of interest shown in the œsophagus in pulmonary tuberculosis. It should, however, be noted as a matter of some significance that the air-filled upper œsophagus, if deviated, may be visible in a plain chest radiograph. It can be confused with other shadows, since the trachea, herniation of the lung across the mediastinum, and cavitation may produce a somewhat similar appearance. Chest radiographs from 200 patients in a sanatorium with, for the most part, moderately advanced pulmonary tuberculosis were examined, and from them twenty were selected which showed deviations of the trachea and mediastinum. Examination by barium swallow showed abnormalities of the œsophagus in many, similar to those described by Tanturri and Lucioni (1934). Others showed a normal course despite marked deviation of the trachea. Four were particularly noted, as it was thought it might be possible to see the air-filled upper œsophagus in the plain chest film. A barium swallow confirmed this.

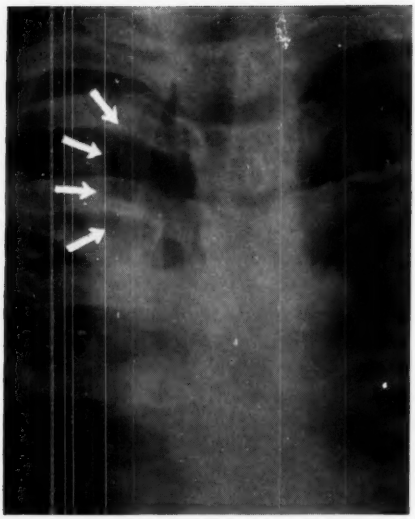

Fig. 1a.—Deviation of the upper œsophagus to the right. Œsophageal wall indicated by arrows.

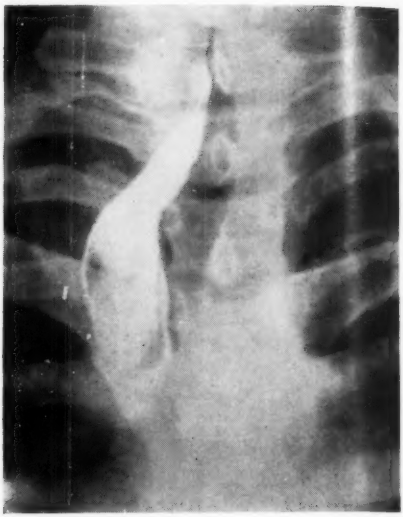

Fig. 1b.—The same after barium swallow.

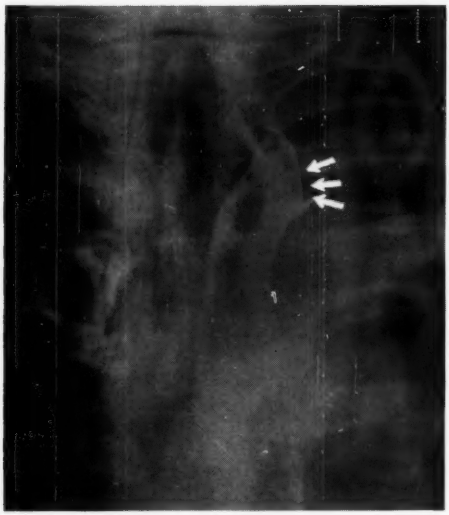

Fig. 2.—Deviation of the upper œsophagus to the left. Œsophageal wall indicated by arrows.

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In these four cases the wall of the œsophagus could be seen as a curved line extending in an arc beyond the wall of the trachea at the level of the second, third and fourth thoracic vertebræ. Between the wall of the œsophagus and the trachea the air-filled upper œsophageal lumen could be seen as an area of increased density and, in one case, the opposite wall of the œsophagus could be seen as a shadow through the lumen of the trachea. Screening showed that the main displacement was lateral. In every case the trachea was displaced, in three to the left and in one to the right, trachea and œsophagus being displaced in the same direction. That the direction of the displacement of trachea and œsophagus is not necessarily always the same is shown by a further case in which the œsophagus was slightly deviated to the left, its air-filled lumen being just visible in the plain chest radiograph, while the trachea was markedly deviated to the right.

All four patients with marked upper œsophageal deviation showed gross pulmonary tuberculosis which had been present for more than six years. None had had symptoms referable to the œsophagus at any time. The three who showed deviation of the œsophagus to the left had all had artificial pneumothoraces for from two to four years. As soon as these were abandoned and the lung allowed to expand, the gradual outward movement of the œsophagus could be seen in the plain radiographs, as well as deviation of the trachea and mediastinum to the left. The patient with deviation of the œsophagus to the right had had pulmonary tuberculosis for fourteen years at the time of examination, showed tracheal and mediastinal displacement to the right, and her disease was mainly right-sided. A right artificial pneumothorax had been induced at the onset of her illness, but was abandoned after a short time. Unfortunately, in the only radiographs available, the œsophageal displacement was already established.

Deviation of the œsophagus in these cases appears to be due to adherence of lung and pleura to the œsophageal wall. The contracting lung has drawn the unsupported upper œsophagus across, leaving unaffected that part related to and supported by the aortic arch. The sharp kink in the œsophagus, the independence of tracheal and œsophageal deviation, and the fact that sudden and reversible deviations of the œsophagus are not found, make it unlikely that the œsophageal displacement is simply a herniation of the œsophagus due to pressure changes on one or other side of the mediastinum.

Summary

Four cases of deviation of the upper œsophagus are described, three to the left and one to the right, occurring without symptoms in long-standing cases of pulmonary tuberculosis with fibrosis of the lung. This deviation of the air-filled œsophagus is visible in the plain chest radiograph. It is important to realise this possibility, as confusion with other shadows may occur. The confusion can be resolved by a barium swallow examination.

My interest in this appearance was aroused by a similar case which Dr. J. M. Battersby pointed out at Papworth. I am indebted to Drs. E. H. Hudson and K. M. A. Perry for permission to record their cases, and to Mr. E. W. Groves, M.S.R., for much help.

One patient has died since this paper was written. Post-mortem examination revealed gross fibrosis and cavitation of the left lung, while the right was herniated anteriorly across

the mediastinum. The left wall of the thoracic œsophagus above the aortic arch lay about 2 cm. to the left of the midline. A thin adhesion connected œsophagus and apical pleura, and an adjoining para-œsophageal lymph node showed microscopic evidence of tuberculosis. Contraction of the neighbouring pleura had drawn the œsophagus across without any dense adhesions forming. There was no sign of tuberculosis of the œsophagus itself.

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CHRONIC PULMONARY HEART DISEASE

By Samuel Oram

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Pulmonary heart disease may be defined as embarrassment and eventual failure of the right ventricle resulting from disease of the lungs or disorder of the pulmonary circulation. Some degree of pulmonary hypertension is invariably present.

The term "cor pulmonale," originally introduced by McGinn and White (1935) to apply to examples of acute right-sided heart failure resulting from pulmonary embolism, is sometimes used as a synonym for pulmonary heart disease whatever the mode of onset or cause of the latter. Its scope has become so wide that there is probably little value in retaining this term, although it has the merit of brevity.

Etiology

Although this article is concerned only with the chronic type of pulmonary heart disease, some of the subacute forms may, on rare occasions, give rise to chronic pulmonary heart disease. A provisional classification is appended (Table I).

Table I.—Classification of Causes of Chronic Pulmonary Heart Disease

1. *Secondary to Lung Disease* (*Pulmonary Anoxia*).
   - (i) Chronic emphysema with or without bronchitis.
   - (ii) Other primary diseases of lung leading to emphysema—pneumokoniosis (silicosis especially), bronchiectasis, fibrocaseous tuberculosis, asthma and congenital cystic lung.
   - (iii) Severe kyphoscoliosis leading to collapse and emphysema ("failure of the hunchback").

2. *Lesions of the Pulmonary Arterial Tree* (*Pulmonary Hypertension*).
   - (i) Primary pulmonary hypertension.
   - (ii) Diseases of the pulmonary aterioles—polyarteritis nodosa, schistosomiasis, amyloidosis, sarcoidosis, scleroderma.
   - (iii) Pulmonary artery lesions—aneurysm of pulmonary artery, obstruction of pulmonary artery or conus by aortic aneurysm.

3. *Following Subacute Pulmonary Heart Disease.*
   - (i) Miliary secondary carcinomatosis of the lung.
     - (*a*) Pulmonary arteriolar embolism.
     - (*b*) Pulmonary arteriolar thrombosis secondary to perivascular lymphatic infiltration.
   - (ii) Repeated pulmonary emboli ("packed emboli") or single organised massive pulmonary embolus.

By far the most important factor which gives rise to chronic pulmonary heart disease is chronic obstructive emphysema. This is true whether it be

associated with the usual recurrent bouts of infective bronchitis or asthma, or whether it be of the less common " compensatory " type associated with extensive fibrosis of the lung from conditions listed in Table I. Even in the kyphoscoliotic examples of pulmonary heart disease it is the emphysema that really causes the right heart to fail and which results from the collapse and fibrosis which in turn are caused by the chest deformity. In some of these kyphoscoliotic examples it is possible that the severe chest deformity causes torsion of the heart, with kinking of the pulmonary artery or its main branches, and this leads to obstruction to the flow of blood from the right ventricle.

Although chronic obstructive emphysema is the commonest cause of pulmonary heart disease, it must be admitted that there is a distinct lack of correlation between the degree of emphysema and the accompanying cardiovascular changes. This was originally pointed out by Parkinson and Hoyle (1937) and more recently confirmed by Brooks (1947).

Although emphysema is the important factor in producing pulmonary heart disease, there is a second, but much smaller, group of cases where there is no demonstrable disease of the lung parenchyma and yet the pulmonary artery pressure is very high—in fact, usually higher than in those cases produced by emphysema. In this group the disorder appears to affect primarily the pulmonary artery or its branches. Sometimes no disease of the pulmonary vascular tree can be demonstrated at autopsy. On the other hand, the pulmonary arterioles may show evidence of specific diseases such as polyarteritis nodosa (Eskelund, 1943), or bilharzia may cause pulmonary endarteritis (Bedford *et al.*, 1946). Scleroderma may lead also to obliteration of the small lung vessels. An unusual cause of vascular obliteration is recurrent pulmonary embolism, and these emboli may organise and occlude the smaller branches of the pulmonary artery (Castleman and Bland, 1946). Even more rarely a main branch of the pulmonary artery may be occluded in an acute episode, and the clot may subsequently organise and perhaps the artery may partially recanalise with the production of chronic pulmonary heart disease.

Even more rare than disease of the pulmonary arterioles are lesions of the pulmonary artery itself which can lead to pulmonary heart disease. Such lesions include aneurysm of the pulmonary artery, or obstruction of that vessel or the conus of the right ventricle by pressure from without of an aortic aneurysm.

A brief mention of the so-called Ayerza's disease must be made here. It is often implied that it is a special syndrome resulting from disease of the pulmonary arterioles, usually syphilitic. Ayerza himself did not claim that his single case of " cardiacos negros " was syphilitic, and in fact he did not describe the pulmonary artery. Brenner (1935) analysed the published cases carefully and concluded that they were nothing more than cases of chronic pulmonary heart disease. There would appear to be no good reason for retaining the term.

It is not only of academic importance to distinguish the two groups of pulmonary heart disease—namely, those resulting directly or indirectly from chronic obstructive emphysema and those resulting from disease of the pulmonary vessels—because they have striking clinical differences and their treatment is different (Table II). Wood (1950) has suggested the terms

TABLE II.—CLINICAL TYPES OF PULMONARY HEART DISEASE.

	Pulmonary Anoxia	*Pulmonary Hypertension*
1. Essential pathology ..	Chronic obstructive emphysema	Disease of pulmonary arterial tree
2. Frequency	Common — 10% of all heart disease	Rare
3. Age	75% are over 50	All ages including childhood
4. Sex	5 males to 1 female	Probably equal (? females commoner)
5. Cyanosis	Central	Peripheral
6. Polycythæmia and clubbing	Usually present	Usually absent
7. Clinical signs of R.V. hypertrophy	May be completely masked by emphysema	Usually obvious
8. Pulmonary infection ..	Common	Absent
9. Radiology	Emphysema invariable	No emphysema
10. P.A. and R.V. pressure ..	Moderately increased	Greatly increased—55-80 mm. Hg
11. Arterial oxygen saturation	Reduced to 80-60% or lower	Normal until preterminally, when may fall to 80%
12. Oxygen therapy	Very beneficial	Useless
13. Effect of digitalis	Sometimes disappointing	Beneficial
14. Venesection	May be dangerous	Beneficial

" anoxic pulmonary heart disease " for the common emphysematous group and " hypertensive pulmonary heart disease " for the smaller vascular group.

CLINICAL PICTURE

In the common type of pulmonary heart disease resulting directly or indirectly from chronic obstructive emphysema the clinical problem is to separate three groups of symptoms and signs—namely, those resulting from the primary disease of the lungs, those which are produced by hypertrophy of the right ventricle and pulmonary artery, and those which are a sequel of failure of the right side of the heart. Emphysema is itself very difficult to diagnose in the early stages, and it notoriously masks other abnormal physical signs both in the lungs and heart. In addition the signs resulting from emphysema alone can closely mimic those of right-sided heart failure and the early detection of heart failure resulting from emphysema may be extremely difficult. In both emphysema and right-sided heart failure dyspnœa is present. Central cyanosis accompanies both. Cough is commonly present and hæmoptysis may occur. Distension of the jugular veins is commonly seen in emphysema and does not necessarily signify failure of the right side of the heart. The exact cause of the cervical venous extension in emphysema is not known. Fishberg (1940) suggests that as the venous pressure is often normal in emphysematous subjects with bulging and seemingly engorged cervical veins, the presence of these vessels may be due to thickening of the wall and dilatation resulting from repeated engorgement during paroxysms of coughing. Kountz *et al.* (1936) have shown that this venous distension in the neck is due to the raised intrapleural pressure of emphysema: the intrapleural pressure may become positive in emphysema and thus increase the resistance to the venous return to the heart. In both conditions the liver may be palpable. In emphysema this does not necessarily imply heart failure, because the liver may

be displaced downwards by the low diaphragm. However, if a hepato-jugular reflux is present, or the liver is tender, it is good evidence of early failure of the right side of the heart. A condition which is more common than often suspected in patients with emphysema is angina hypercyanotica, which can very closely simulate angina pectoris. It can occur in both emphysema alone or with accompanying failure. Recently Viar and Harrison (1952) have suggested the term " pulmonary hypertensive pain," as cyanosis is commonly absent in these patients except during the episodes of pain, although the pulmonary artery pressure remains high. The severe bouts of coughing sometimes associated with emphysema and bronchitis may lead to temporary loss of consciousness. This was originally described as laryngeal vertigo by Charcot (1876). Sometimes these cases are wrongly diagnosed as having cardiovascular disease because of the loss of consciousness, but Baker (1949) has shown that they are common in emphysema in the absence of heart disease. He suggests that the attacks are due to prolonged increased intrathoracic pressure against the closed glottis, resulting from violent coughing. This leads to diminished venous return to the right heart with consequent reduction in its output and reduction of the blood flow to the left heart chambers. The reduced stroke volume of the left ventricle and reduced systemic pressure impairs the cerebral circulation, causing faintness or unconsciousness. The rise in intrapleural pressure with coughing in emphysema aggravates the above effect. Most authorities agree that the presence of œdema of the ankles associated with emphysema, provided other causes are excluded, is good evidence of failure of the right side of the heart, but Kountz and his co-workers (1936) believe that even this may result from emphysema without heart failure, the anoxia increasing the capillary permeability.

Signs of Pulmonary Fibrosis and Emphysema

The so-called barrel-shaped chest is often taken as an indication of emphysema, but it commonly occurs in the absence of that disease. Diminished expansion is usual in emphysema, and may be localised in unilateral fibrosis. Diminished tactile fremitus and a hyper-resonant percussion note with diminution or obliteration of the cardiac dullness result from emphysema. On auscultation, faint breath sounds, often with prolonged expiration, portray a loss of elastic tissue. An infective element gives rise to scattered râles and rhonchi, which thus do not necessarily signify heart failure. Cyanosis is common in moderate emphysema, with or without fibrosis, but if it is very intense it, in the author's opinion, suggests that the heart has begun to fail and may provide a useful early indication of cardiac involvement. Clubbing of the fingers and toes, and polycythæmia, correspond roughly to the degree of cyanosis. Baldwin *et al.* (1939), by means of cardiac catheterisation, claim that polycythæmia associated with emphysema is evidence that the heart has become involved. It was absent in their cases of emphysema without heart failure, regardless of the severity of the arterial anoxia.

Signs of Right Ventricular Hypertrophy

The signs of an enlarged right ventricle are few and are often completely masked at the bedside by the accompanying emphysema. A manœuvre which

the author has found useful is to place the middle finger of the right hand on the third left costal cartilage, the index finger in the second intercostal space above and the ring finger in the third intercostal space below. The middle finger, which overlies the pulmonary valve, may then feel the closure of that valve if pulmonary hypertension is present and thus indirectly right ventricular hypertrophy may be suspected; the index finger overlying the pulmonary artery may then appreciate pulsation of that vessel if it is distended; the ring finger may feel pulsation of the conus of the right ventricle, and if it is moved down to the fourth intercostal space near the sternum the impulse of an hypertrophied right ventricle may be appreciated. The impulse of the apex in right ventricular hypertrophy is often of a more sudden character than the heaving or sustained cardiac impulse of left ventricular hypertrophy, giving rise to a sensation sometimes described as tumultuous. However, in emphysema the cardiac impulse is commonly impalpable and in any case, as will be seen later, left ventricular enlargement is commonly found in these patients with emphysema. Percussion of the left border of the heart is even more unprofitable than usual in the presence of emphysema. Auscultation in the presence of pulmonary hypertension may reveal a pulmonary systolic murmur and a loud pulmonary second sound which is commonly split, the pulmonary element of the split sound being the louder. A third sound may be audible too.

Signs of Right Ventricular Failure

As mentioned previously, the detection of early right ventricular failure in the presence of emphysema may be very difficult. The earliest sign is probably a positive hepato-jugular reflux. To elicit this the patient is sat upright. Normally no distension of the cervical veins is seen, although in healthy individuals one vein, especially the left, is occasionally distended for no apparent reason. If the reflux is present pressure over the hepatic region causes distension of the cervical veins from below whilst the pressure is maintained. Commonly the liver is found to be tender too, and some say that the hepatic tenderness is even earlier than the reflux. Distension of the jugular veins is not in itself an indication of right ventricular failure, or for that matter even necessarily an indication of a raised venous pressure—it is common in uncomplicated emphysema for reasons mentioned earlier.

Œdema of the ankles in the absence of such other causes as varicose veins, flat feet, anæmia and so forth is usually considered good evidence of right ventricular failure. A protodiastolic triple rhythm in an adult over forty is good evidence of failure of the right ventricle, although the heart sounds are so often obscured by emphysema. When frank congestive failure sets in deep cyanosis and generalised œdema with ascites and pleural effusions will be evident, but, as shown by Parkinson and Hoyle (1937), death from congestive heart failure in emphysema is decidedly uncommon. In some cases, but not all, the cardiac failure is of the high output type. Thus, although the patient's hands are blue they are warm, and the pulse volume is full and of the collapsing variety, and capillary pulsation may be evident. Papillœdema may be present too. The cyanosis is central and detectable in warm situations such as the conjunctivæ or buccal mucous membrane.

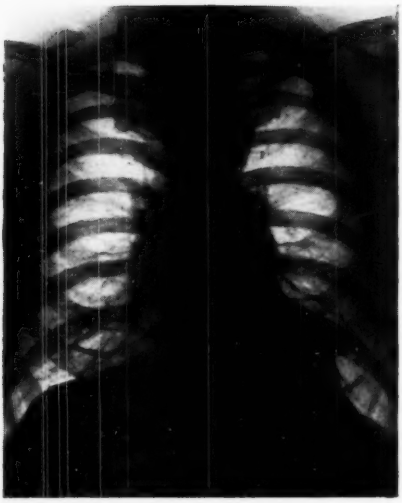
The small group of cases of pulmonary heart disease in which the pul-

monary arterial tree is primarily affected and emphysema is absent, called by Wood (1950) hypertensive pulmonary heart disease, shows certain differences clinically from the commoner group due to chronic obstructive emphysema. The hypertensive cases may occur, although rarely, in childhood, and the cardiac output is then low and the cyanosis peripheral, so that the extremities are cold. The findings are usually those of pure right ventricular failure with normal rhythm, although auricular flutter and fibrillation do occur from time to time. Because of the absence of emphysema in this group the physical signs of right ventricular hypertrophy are much more obvious. A loud pulmonary second sound with exaggeration of the pulmonary element is present and a protodiastolic triple rhythm may be heard. The jugular venous pressure is raised and the " a " wave is conspicuous. The liver is enlarged and tender and functional tricuspid incompetence may be present. In spite of gross signs of right-sided heart failure there is no orthopnœa and the lungs are dry. According to Wood (1950), the pressure in the right ventricle and pulmonary artery is usually extremely high, the mean pulmonary artery pressure in his cases was 55-81 mm. Hg.

Investigations

Radiology.—The demonstration radiologically of even quite severe emphysema does not, of course, imply the presence of pulmonary heart disease. Radiology is not of great value in detecting early cardiac involvement. Emphysema results in the diaphragm being low and flattening of the curves of the cupolæ. Sometimes each cupola has a scalloped appearance. On fluoroscopy the diaphragmatic excursion is seen to be diminished. The rib spaces are widened, the ribs run more horizontally than usual and they tend to be crowded at the apices (" tiling "). In the antero-posterior view the lower part of the chest wall is often indented laterally. The lung fields are excessively radiolucent, especially in their lower part, and the faint outlines of bullæ may be seen. The translucent lung fields help to explain the heavy hilar shadows, which therefore do not necessarily imply early heart failure. The low diaphragm results in elongation and clockwise rotation of the heart around its longitudinal axis as looked at from below, the cardiac size being thus deceptively small—the so-called " ribbon," " strap " or " tropfenherz."

The radiological evidence of hypertrophy of the right ventricle is usually scanty and the " cœur-en-sabot " appearance is not seen. Parkinson and Hoyle (1937) showed that although when looked at in the antero-posterior view the heart in advanced emphysema may appear to be smaller than normal, by rotating the patient into the oblique positions the pulmonary conus will be found enlarged in 40 per cent of cases, and of these a half will also have radiological evidence of enlargement of the right ventricle. Later, when the inflow tract of the right ventricle hypertrophies, the right ventricular enlargement is best seen in the left anterior oblique position (Schwedel, 1948). The curve of the pulmonary artery may be a little prominent and is often combined with that of the conus. On rare occasions the pulmonary artery curve may be seen to become exaggerated very rapidly, almost into aneurysmal proportions (Wood, 1950). Although prominence of the main branches of the pulmonary artery at the hila, with or without dilatation of the main pul-



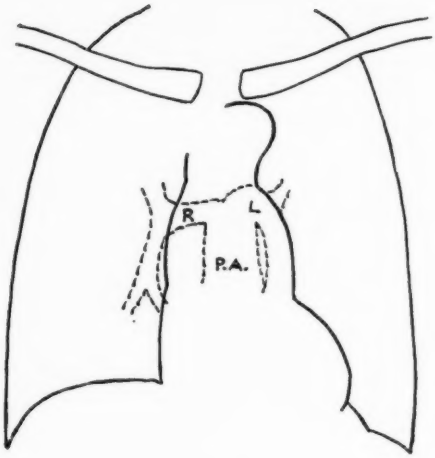


Fig. 1.—Antero-posterior view of heart showing how the descending branch of the left pulmonary artery can be mistaken for the so-called "middle arc" which is usually formed by the main trunk of the pulmonary artery (P.A.). The left pulmonary artery (L) is commonly slightly higher than the right (R), as shown.

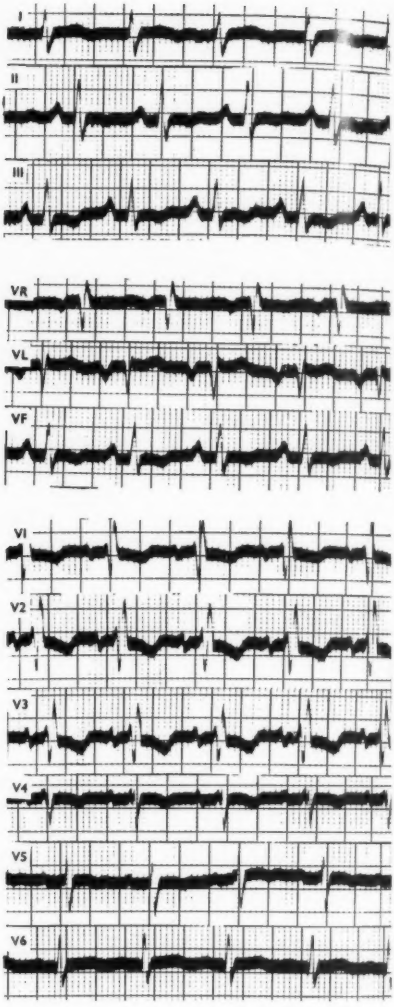


Fig. 2.—Cardiogram from a case of chronic pulmonale resulting from severe bronchitis and emphysema with asthma of 10 years' duration in male aged 28. B.P. normal. No digitalis given. Sinus rhythm. Typical P Pulmonale in Leads III and VF. RS-T segment depression and inversion of the T wave in Leads II, III, VF and V1 to V5 indicative of right ventricular hypertrophy. The tall R wave in V1 also suggests right ventricular hypertrophy. Transitional point between V5 and V6. Intrinsicoid deflection delayed in V1 to 0·05 sec. (see text).

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monary trunk, is present in more than half the cases of severe emphysema, the changes are rarely conspicuous until pulmonary heart disease is advanced and then there may be some pulsation of these vessels visible at fluoroscopy. The right auricle becomes distended when failure supervenes and forms a prominent convex bulge above the diaphragm in the antero-posterior view, and the upper mediastinal shadow may then be widened owing to distension of the superior vena cava. Pleural transudates may finally appear.

Systemic hypertension is not uncommonly found, together with pulmonary heart disease, and this often modifies the radiological appearances. If the patient's blood pressure is normal, however, a prominent and vigorously rocking aortic knob should suggest a high cardiac output. Notwithstanding the common occurrence of hypertension or coronary artery disease in patients with pulmonary heart disease, many examples are met with where there is definite enlargement of the left ventricle without satisfactory explanation. It is possible that some of these otherwise inexplicable examples of left ventricular enlargement are due to the persistence of a high cardiac output.

Angiocardiography

Recent studies have indicated that at least in some cases the prominence of the so-called " pulmonary conus " is due to enlargement of the pulmonary trunk and not to the conus of the right ventricle. Sometimes the descending branch of the left pulmonary artery can simulate enlargement of its main trunk, especially if insufficient penetration is used radiologically (Fig. 1). In the early stages of right ventricular hypertrophy, undetectable by ordinary radiology, angiocardiography may show that the ventricular septum is bulged to the left instead of to the right as in the normal.

Electrocardiography

Although emphysema alone can influence the electrocardiogram in so far as it influences the position of the heart, and although occasionally in the presence of undoubted pulmonary heart disease the electrocardiogram may be normal, nevertheless the cardiogram is capable of yielding one of the earliest signs that the heart is being affected by pulmonary heart disease (Fig. 2). In the author's opinion it is of considerably greater value in early diagnosis than radiology and is only second to cardiac catheterisation.

Because the heart is elongated as a result of the low diaphragm, it tends to be vertical electrically. This is shown by a tall R wave in unipolar limb lead VF and often by a deep S wave in lead VL. If the heart is very vertical VR and VL may be indistinguishable. The complexes in leads representing electrical activity of the left ventricle, such as V_5 and V6, resemble the configuration of lead, VF, because the apex of the heart tends to point towards the left leg rather than the left arm. In the standard leads this is represented by either a small R wave in Lead I and a tall R wave in Lead III or by right axis deviation—namely, a deep S wave in Lead I and a tall R wave in Lead III. The clockwise longitudinal rotation, which may also result from emphysema uncomplicated by right ventricular enlargement, causes the transitional point, which approximately marks the position of the ventricular septum and is the point on the chest leads where the R and S waves are of

the same amplitude, to be displaced to the left. Instead of appearing between leads V4 and V5 as in the normally positioned heart, it is displaced beyond V5 or even V6.

When the right ventricle hypertrophies the so-called " right ventricular strain " appearance may be seen—namely, depression of the RS-T segment with inversion of the T wave in leads overlying the right ventricle, such as V1 and V2. In these leads the intrinsicoid deflection will be later than normal because the right ventricular wall is thicker. The intrinsicoid deflection in V1 is normally delayed for 0·02-0·03 sec., but in right ventricular hypertrophy it may be delayed to 0·04-0·05 sec. Very good evidence of hypertrophy of the right ventricle is the appearance of a tall R wave in lead V1 and occasionally in V2 also.

When the right ventricle is embarrassed this is soon reflected by distension of the right auricle, and it is probably this which results in the P waves being tall and " spiky " (P pulmonale), particularly in lead VF and standard leads II and III. Wood (1948) has shown convincingly how important and early is this sign, which was present in 85 of his 100 cases. It is probably the earliest sign that the lung disease is affecting the heart, or at least as early as elevation of the diastolic right ventricular pressure (see below). According to Wood the normal P wave rarely exceeds 1·5 mm. in amplitude and averages 1 mm. The P pulmonale is commonly 2 or 3 mm. tall, but is never widened. Its exact causation is obscure—it is not due to anoxia or an increased cardiac output and probably results from an increase in right auricular pressure.

Catheterisation Studies

These have rendered it possible to offer a tentative account of the pathological physiology in pulmonary heart disease. In the usual type of cor pulmonale obstructive emphysema causes obliteration of a large part of the pulmonary vascular bed and this increases the resistance against which the right heart must work. The consequent dilatation and hypertrophy proceed backwards as it were, involving in order the pulmonary artery, conus, right ventricle and lastly the right auricle. Bloomfield *et al.* (1946) found that although patients with emphysema but no right-sided heart failure frequently showed elevation of the ventricular systolic pressure, the ventricular diastolic pressure in these patients was normal and so was the pressure in the right auricle. Once right-sided heart failure set in the ventricular diastolic pressure and the right auricular pressure rise, and this increase is almost certainly the earliest sign of pulmonary heart disease.

The pulmonary hypertension leads to atheroma of the pulmonary artery and arteriosclerotic intimal thickening of the pulmonary arterioles, and once these physiological changes have occurred they themselves may further aggravate the pulmonary hypertension by increasing still more the obstruction to the outflow of the right ventricle. Once anoxia has been produced it too can probably directly increase the pulmonary artery pressure.

The decreased pulmonary capillary bed not only leads to a rise in the pressure in the pulmonary circuit but also causes deficient aeration of the blood flowing through the lungs. The arterial oxygen saturation therefore falls and by arterial puncture is usually found to be 60-80 per cent. Another cause of this fall in the arterial oxygen saturation is incomplete mixing of the air in

the lung, so that even the alveolar air may contain an abnormally low oxygen content. The loss of pulmonary elasticity greatly reduces the vital capacity, usually to less than 1,500 ml., and this causes a reduction in the maximum breathing capacity (*i.e.*, the maximum volume of air that can be ventilated per minute). To compensate for this the minute volume of respiration (*i.e.*, the volume of air the patient can actually ventilate per minute) rises above normal and the patient therefore has to do more work to abstract the required oxygen from the inspired air.

Because the arterial oxygen saturation falls the cardiac output increases to maintain adequate oxygen to the tissues. McMichael and Sharpey-Schafer (1944) have shown that in chronic pulmonary heart disease the cardiac output is usually moderately raised to 5-9 litres per minute. Secondary polycythæmia, stimulated by anoxia of the bone marrow, is also compensatory.

Estimation of the Circulation Rate

Although theoretically attractive, such estimations to be of value must be taken serially, and many of the present techniques do not yield a sharp end point, but perhaps with the increasing use of radio-active isotopes this will be solved for the clinician. However, Gillianders (1949) is of the opinion that serial circulation times are of considerable value in determining the onset of failure in patients with emphysema.

Treatment

There is no doubt that chronic bronchitis and emphysema are still often regarded lightly, almost casually. Prophylactically, avoidance of recurrent attacks of infection, which are so common in emphysema, is important. Unfortunately this is often a question of climate and is commonly not feasible. Continual coughing as a result of cigarette smoking should be avoided.

Once signs have appeared they should be taken seriously. Bronchial spasm if severe will need adrenalin intramuscularly. If not so severe, then ephedrine or nor-adrenalin orally may suffice. Occasionally ephedrine causes insomnia, but in my experience it more often gives the patient a better night by relieving his dyspnœa. Unfortunately it may cause frequency of micturition or even retention of urine in the elderly. Isopropyl-nor-adrenalin is given as a 20 mg. tablet sub-lingually and can be repeated several times a day. The anti-histaminics are not a substitute for more powerful anti-spasmodics.

Infection must be dealt with early and, rather than awaiting the results of sputum culture and bacterial sensitivity tests, a sulphonamide or anti-biotic with a wide range should be employed. Of the wide range of relatively non-toxic sulphonamides sulphafurazole (Gantrisin, Roche) is promising, and the average dose is 2 grammes followed by a gramme six-hourly for a few days. Chloramphenicol orally in doses such as two capsules each of 250 mg. given six-hourly for a few days is also of great value.

If there is deficient expansion of the chest a course of breathing exercises may help, but when cyanosis is severe, then, provided it is central in origin, as it usually is, the beneficial effect of an oxygen tent is often dramatic. Oxygen therapy is useless in the rare cases of idiopathic pulmonary hypertension with peripheral cyanosis.

The value of digitalis has been questioned and it has even been considered harmful if not dangerous. Sudden death is said to result from its use. Although it is true that patients with severe right-sided heart failure secondary to lung disease are prone to sudden demise, I have never been convinced that this results from digitalis. Far from it, in the presence of failure it should always be tried cautiously, and often the response is most gratifying.

Theophyllin ethylene-diamine (Cardophylin, Whiffen) in addition to its bronchodilator effect, also dilates the coronary arteries if given intravenously and may help to tide over an emergency. It is given into the vein slowly and well diluted in amounts such as 0·24-0·48 G. I have never been impressed by its action by mouth.

If the patient remains in a state of chronic right-sided heart failure (" obstinate failure ") in spite of the above measures, then thiouracil should be seriously considered in an attempt to lower his oxygen consumption.

Mercurial diuretics, with or without a low sodium diet, have been condemned in chronic pulmonary heart disease. Unfortunately the use of Mersalyl, by lowering the venous pressure, may reduce the raised cardiac output which, as already explained, is part of a compensatory mechanism for the low respiratory gaseous exchange. Howarth, McMichael and Sharpey-Schafer (1947) have shown that the venous pressure may be already at an optimum level and if it is lowered the cardiac output will fall. However, if generalised anasarca is increasing and mercurial diuretics are withheld the patient will certainly die.

There are two measures which are particularly harmful in this condition. The first is the use of morphia, which depresses the respiratory centre, pethidine being strongly preferable. The second is venesection: there are objections to venesection similar to those against mercurial diuretics, but, as the action of venesection is so sudden, if its indications have been misjudged it may cause rapid death.

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ENLARGED HILAR AND PARATRACHEAL GLANDS FOLLOWING B.C.G. VACCINATION

By L. D. Richards and L. Steingold

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B.C.G. vaccination has been used for a sufficiently long time and on a sufficiently large scale to have established, beyond doubt, its harmlessness. Significant reactions are infrequent and are generally of a local nature. Thus Weill-Hallé and his colleagues (1949) report that vaccination by the subcutaneous route produced local erythema and œdema in 8·5% of cases and local abscess or ulceration, which healed readily, in 0·9%. Vaccination by scarification produced axillary or cervical adenitis, which followed a benign course, in 0·3% of cases. Difs (1947) reported that the commonest severe complication occurring in his experience was the production of local necrosis in 0·3% of cases.

Nevertheless, occasional reports have drawn attention to more generalised lymphadenopathy and other reactions to the vaccination. Le Melletier *et al.* (1950) report asymptomatic mediastinal lymphadenopathy in two infants, one vaccinated orally and the other by scarification. Mediastinal gland enlargement was also reported, in adults, by Larsen (1950) in eight cases, three of them within two months of vaccination and two within six months. Of these latter five cases, one showed episcleritis and erythema nodosum in addition and another bilateral iritis. Von Sydow (1950) reported a patient who developed suppuration at the site of vaccination, followed by splenomegaly, a raised erythrocyte sedimentation rate and an abscess of the regional lymph glands. Cultures of an avirulent tubercle bacillus resembling B.C.G. were obtained from the site of vaccination and the abscess of the lymph glands. Roudinesco and Jupeau (1946) observed pains in the joints, fever, stupor and X-ray changes suggesting mediastinal adenitis in one of their cases, and Lecroix *et al.* (1951) have observed fever, adenopathy and pulmonary symptoms in one of their patients.

Such reactions, sometimes alarming when they occur, follow nevertheless a benign course.

We have observed a patient who developed cervical and mediastinal adenitis and pulmonary X-ray changes following B.C.G. vaccination.

The patient, a nurse of 18 years, began her Mantoux tests on October 26, 1949, the usual three strengths being employed. By May 31, 1950, she had been tested nine times, with negative results. B.C.G. vaccination was performed on June 2, 1950, the local reaction to which was a slightly raised red papule, 4 mm. in diameter, appearing on the day following vaccination, and remaining much the same in appearance until ulceration occurred some time between June 28 and July 5, 1950. Gradual healing took place. No glandular enlargement was observed in neck or axilla during this period. The B.C.G. batch number was 896.

Mantoux testing subsequent to the vaccination showed the nurse negative to 1/10,000 on July 19, 1950, but positive to 1/1,000 on July 26, 1950 (erythema 20 mm., induration 15 mm.). A post conversion X-ray (August 2, 1950) showed no abnormality, but a routine X-ray (December 29, 1950) showed bilateral enlargement of hilar and paratracheal glands. No changes in the lung parenchyma were noted in this film.

Eight other nurses had been vaccinated at the same time with the same batch of vaccine. All converted to Mantoux positive, and in August 1951, more than a year later, were still Mantoux positive. Repeated X-rays of their lungs have not shown pulmonary or mediastinal abnormality. No Mantoux was done on the patient until December 1951, when she gave a positive reaction to 1/1,000 O.T.

Following the discovery of the X-ray abnormalities, and although the patient was free from symptoms and apparently well, she was confined to bed. Physical examination revealed no abnormality other than a small palpable gland in the left posterior triangle of the neck. This was subsequently removed for histological examination. Temperature, pulse and respiration were normal and remained so throughout. By the end of March 1951 she was up all day and returned home for convalescence. She returned to London in September 1951 and is on full duty but under observation.

Her erythrocyte sedimentation rate did not rise above 10 mm. (one hour: Westergren) while she was confined to bed. While she was on convalescence it rose to 18 and 20 mm., but had fallen to 4 mm. by July 25, 1951.

Her leucocyte count was 4,150 per c.mm. on January 20, 1951, three weeks after the X-ray showing mediastinal glandular enlargement, 64% being granulocytes, of which about half were metamyelocytes and juvenile forms. Eleven days later, on January 31, the picture was much the same (4,700 leucocytes per c.mm., polymorphs 61·5%, eosinophiles 1·5%, lymphocytes 34·5%, monocytes 2·5%) and the polymorphs showed a similar "shift to the left." A month later, on February 28, the leucocyte count was 5,800 per c.mm., polymorphs 64%, eosinophiles 1%, lymphocytes 32%, monocytes 3%. No metamyelocytes were observed, and the "shift to the left" had now disappeared. The picture was considered normal. Repeated cultures of laryngeal swabs and fasting gastric juice failed to grow tubercle bacilli, and Paul-Bunnell tests were negative.

Histological examination of the cervical gland showed very heavy epithelioid cell reaction in confluent masses throughout the gland parenchyma (Fig. 1) with multinucleate cell formation of Langhans' giant-cell type and a little necrosis of the centres of the cell masses. The specimen was received in formalin and cultures were impossible. No tubercle bacilli could be demonstrated in the sections.

The series of changes in the X-ray films are illustrated (Figs 2-4). The film of March 1951, ten months after her vaccination, was the first to show diminution in the size of the glandular shadows, and in it for the first time a fine generalised mottling was seen in the lower two-thirds of each lung field. In the film of November 7, 1951, the left hilar glands are the only ones showing any enlargement and the parenchymal changes are almost gone. The latest film, March 13, 1952, shows no glandular enlargement and no mottling.

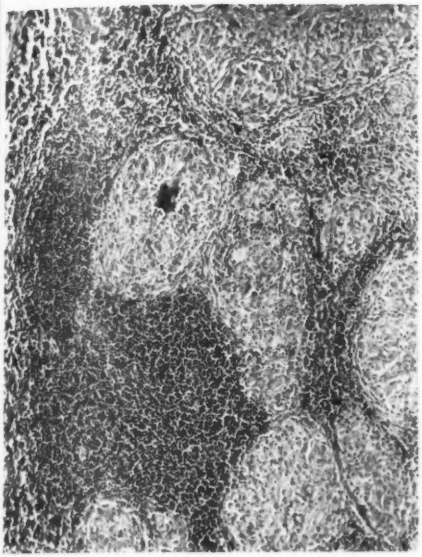

Fig. 1.—Section of cervical gland, showing very heavy epithelioid cell reaction in confluent masses throughout the gland parenchyma, with multinucleate cell formation of Langhans' giant cell type, and a little necrosis of the centres of cell masses. ×65.

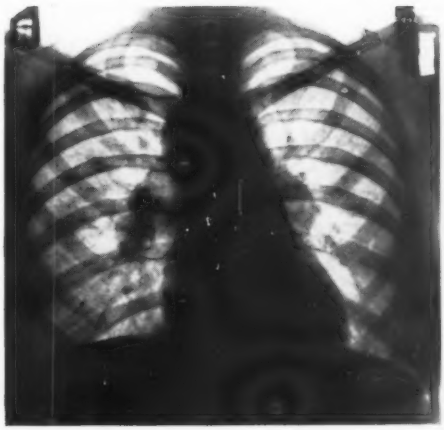

Fig. 2.—Film taken 29/12/50, seven months after B.C.G. vaccination, showing bilateral enlarged hilar glands, and paratracheal gland. No fine mottling visible.

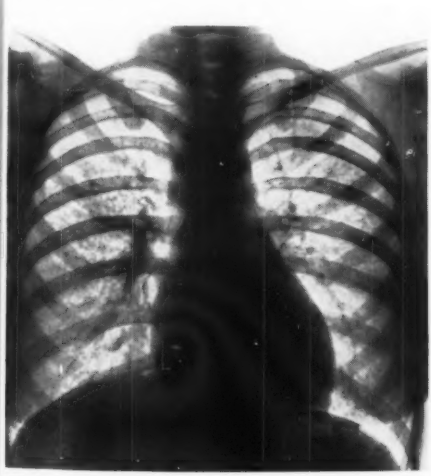

Fig. 3.—Film taken 10/7/51, showing diminution in size of glands. Fine mottling visible in lower two-thirds of each lung.

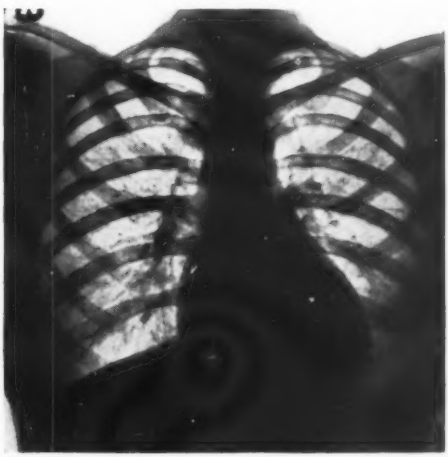

Fig. 4.—Film taken 7/11/51, showing practically complete resolution of glandular swellings and disappearance of parenchymal mottling.

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Discussion

Three possible explanations present themselves in this case. The changes may be due to the B.C.G. vaccination itself. This is the opinion that we hold in view of the very benign course and somewhat unusual picture presented.

The condition may be the result of an exogenous infection with *M. tuberculosus* in a successfully vaccinated subject who had acquired a degree of immunity consequent upon vaccination, sufficient to modify but not to prevent the disease.

And finally, although this is unlikely, it may be a primary infection in a subject who, having being successfully vaccinated, rapidly lost all her immunity before exposure to the source of her primary infection.

The interval of twenty-six days between vaccination and the appearance of ulceration suggests that the nurse was not, at the time of vaccination, in the pre-allergic stage of a natural infection of so recent a date as to allow a negative result to her last (1/100) pre-vaccinal Mantoux test.

Similarly, it is evidence against this being a case in which a natural infection, acquired earlier in life, had by this time become a latent lesion with a return to a Mantoux-negative state, and in which the glandular swelling and parenchymal lesions were the result of reactivation of this latent lesion by B.C.G. vaccination.

There is undoubtedly a close resemblance between the X-ray pictures of our case and those of sarcoidosis reported by Scadding (1950). This is particularly so in the enlargements showing the pulmonary mottling present in both instances. This case may therefore be one of tuberculous sarcoidosis caused by infection with B.C.G.

We do not feel, however, that any satisfactory discussion can be entered into on this point at present.

Summary

A case of cervical, hilar and paratracheal lymphadenopathy, accompanied by radiological lung changes, is reported in a B.C.G. vaccinated nurse. The possible etiology is discussed.

Our thanks are due to Professor Frederick Heaf, Professor of Tuberculosis, University of Wales, for kindly seeing the case and for his many helpful suggestions, and to J. R. M. Whigham, Esq., F.R.C.S., Surgeon Superintendent, St. Andrew's Hospital, Bow, E.3, for removing the cervical gland for section.

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TUBERCULOUS PULMONARY INFILTRATION AS A SEQUEL TO B.C.G. VACCINATION

By Theodore James

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In many parts of the world there is propaganda for mass vaccination of susceptible elements of the populations against tuberculous infection. On the other hand, there is a considerable body of opinion firmly opposed to B.C.G. vaccination as the best means to achieve this end.

Complications directly attributable to B.C.G. vaccination may include any of the following: regional lymphadenitis with or without cold abscess formation, erythema nodosum, phlyctenular conjunctivitis and meningitis. Hlouskova and Houstek (1950) mention three children who developed tuberculous changes in the lungs at seven-day, two-month and four-month intervals after vaccination. But they do not give details of the kind of tuberculous changes which occurred, and nowhere have I seen a description of pulmonary infiltration as in the case presented here.

Case Report

W. D. W., a baby girl aged nine months, was brought to the Out-Patient Department of the Duchess of York Hospital for Babies. The chief complaint was a large swelling in the left axilla which that morning had discharged spontaneously a yellowish-white material. The father, who was alive and apparently well, had, six years earlier, been diagnosed as a case of active tuberculosis, and, for this reason, a regional Chest Physician had given the baby the protection of B.C.G. vaccination.

This was carried out when the baby was about seven months old. The intradermal technique was used, over the left deltoid region. At no time following the vaccination had the baby shown any constitutional upset although the day before attending the hospital the stools had become loose and the baby had vomited once. The mother was well aware of the reason for the vaccination but the regional swelling and the discharge had rendered her entirely out of sympathy with the purpose of the vaccination. This was her only child.

Physical examination showed a very well nourished, healthy-looking baby, in no distress. The initial vaccination was still in evidence and was proceeding along expected lines, but in the left axilla there was a swelling the size of a hen's egg, which was discharging a yellowish-white material—it was a painless cold abscess of the axillary regional lymph nodes. No cervical lymph nodes were palpable. Clinical examination of the chest revealed no abnormality, but a radiological examination disclosed extensive infiltration of the upper lobe of the left lung and a degree of collapse indicated by the cardiac and mediastinal shift to the side of the lesion (see Fig. 1).

Acid-fast bacilli were found in the material from the abscess, but three gastric washings by direct examination and guinea-pig inoculation failed to show the tubercle bacillus. A later swab from the discharging abscess showed polymorphonuclear leucocytes, some lymphocytes, and on culture grew a *Staphylococcus aureus*, which was sensitive to streptomycin, aureomycin and chloromycetin, but insensitive to penicillin.

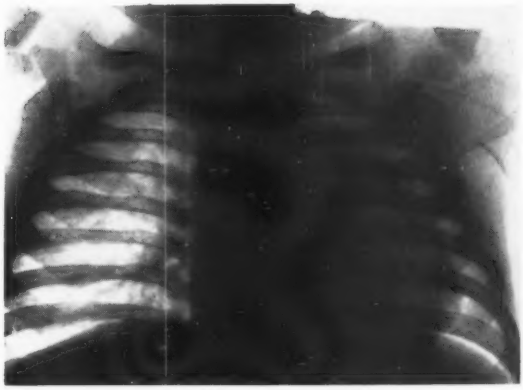

Fig. 1.—Skiagram showing the infiltration of the left upper lobe.

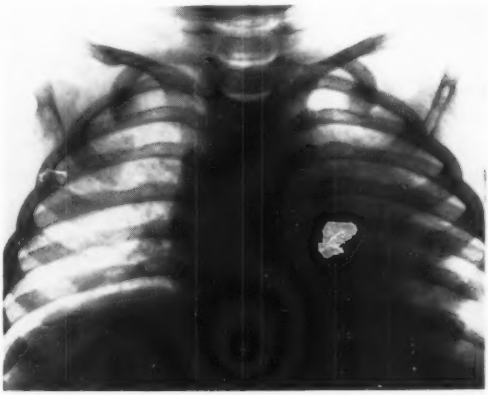

Fig. 2.—Skiagram showing beginning of re-aeration of left upper lobe.

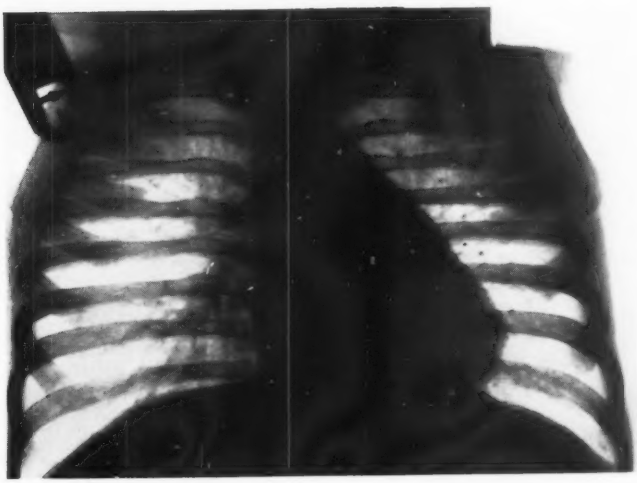

Fig. 3.—Skiagram showing complete re-aeration of left upper lobe.

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Course.—The child continued asymptomatic except for the regional ulcer, which after six months had still not healed completely despite systemic and local streptomycin and para-aminosalicylic acid. Repeated chest X-rays showed no change until six months after the initial examination, when, for the first time, the infiltration of the left upper lobe showed signs of clearing and re-aeration, with a shift of the heart and mediastinum towards their normal positions (see Fig. 2).

At the time of writing—that is, eight months after the first attendance—the skiagram of the lungs shows complete resolution (see Fig. 3).

Comment

This case of pulmonary infiltration, a sequel to B.C.G. vaccination, is exactly comparable with the cases which Martin (1932) included in her clinical group III series of tuberculous pulmonary infiltration, as it occurred in the children which she investigated; that is to say, a symptomless infiltration without physical signs was detected on a routine X-ray examination suggested by the local B.C.G. cold abscess formation in the lymph nodes of the axilla, which drained the lymph from the site of vaccination.

The absence of clinical signs in the lungs, the characteristic radiological picture and the course followed were typical of Eliasberg's so-called epituberculosis, a first infection of the lungs by tubercle bacilli. The absence of pulmonary distress and constitutional upset of the least degree excludes other causes for the pulmonary infiltration.

The acuity or absence of symptoms in this type of lung pathology contracted by contact with an infective person appears to depend largely upon such variable factors as the dosage, virulence of the infecting bacillus, the frequency of such infection and the nature of the patient's reaction. In the case presented here, these factors were 0·5 mg. of B.C.G. introduced on one occasion intradermally in a robust infant, who was anergic to tuberculin. Its significance lies in the involvement of the lymphatic system following infection of the skin, and in the upper half of the body this involvement may be sufficient to produce the pathological process usually attributable to infection by tubercle bacilli within the respiratory system. It remains, however, uncertain whether this pulmonary complication of B.C.G. vaccination was the result of direct lymphatic spread from the vaccinated arm to the pulmonary lymphatics or indirectly due to pulmonary lodgement of the bacilli after hæmatogenous dissemination throughout the body. By virtue of the fact that B.C.G. vaccination is followed by lymphogenous and hæmatogenous spread both routes of systemic infection appear possible.

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A NEW CONTRAST MEDIUM FOR BRONCHOGRAPHY

WITH A COMPARISON OF IODISED OILS AND UMBRADIL *VISCOUS B*

By J. Spencer Jones

From the Royal Northern Hospital, London

Introduction

It is thirty years since Forestier and Leroux added bronchography to the diagnostic aids available in the investigation of chest disease. During this period iodised oils have been universally used as the radiographic contrast medium.

In 1948 Morales and Heiwinkel published a preliminary communication concerning a new contrast medium called Umbradil. Despite further reports in foreign literature no comment has appeared in this country.

It is the purpose of this article to review the value of iodised oils in bronchography and to determine the position at present occupied by the new substance Umbradil.

Ideal Properties for Contrast Media

These may be listed:

(i) No toxic side effects.
(ii) No irritant effects upon the lungs or bronchi.
(iii) Good contrast properties.
(iv) Ability to delineate all bronchi irrespective of size. (Delineation is preferable to filling.)
(v) Alveolar filling should be difficult to achieve.
(vi) The medium should be rapidly removed from the lungs and bronchi.
(vii) The procedure should be well tolerated by the patient.

Iodised Oils

These media possess very good contrast properties. Irritant effects upon the bronchi are minimal, and any alveolar changes which occur (Brown, 1928) probably do not differ from those obtained when physiological saline is introduced into the bronchi. Toxic side effects are rare and seldom fatal. The author has seen one case of rhinorrhœa and lachrymation in a series of thirty-two consecutive patients specifically questioned.

Iodised oils usually give good delineation of the bronchi, but occasionally " filling " of the bronchi occurs. It is probable that this feature, together with undesirable alveolar patterns, is attributable to the low viscosity of the medium. Cooling of the medium before use may not overcome this difficulty completely, and in some cases thickening agents (sulphanilamide or bismuth subnitrate) have been added with apparent advantage.

The persistence of the medium represents the chief drawback to the use

of iodised oils. This carries two disadvantages. Firstly, it renders the interpretation of subsequent radiograms difficult, and, secondly, the oil may predispose to post-operative pulmonary collapse—*e.g.*, after lobectomy (Belsey, 1937). To a considerable extent this can be avoided by encouraging coughing and posturing immediately after bronchography.

It is to the credit of iodised oils that they are well tolerated by the patient. Provided that technique is good, coughing can almost invariably be controlled when bronchograms are being obtained.

Umbradil *Viscous B*

This is the trade name of a new radiographic contrast medium for bronchography. The radio-opaque material is 3 : 5 diiodo-4-pyridone-N-acetic acid (Umbradil) made soluble by the addition of diethanolamine. Iodine confers opacity upon the compound and the combination is such that iodism cannot occur.

Umbradil is put up in aqueous solution with added carboxymethyl cellulose and xylocaine 0·5 per cent. The former is a thickening agent and the latter a local anæsthetic. The combination is named Umbradil *Viscous B*.

The medium resembles colourless melted gelatine and pours with difficulty, but can be easily expressed from a standard 20-ml. syringe. Although viscosity is marked, surface tension is low and spread to the extremities of the bronchial tree and " delineation without filling " is to be expected.

Toxic effects from Umbradil are very rare and have been attributed to individual hypersensitivity. Contra-indications to its use are respiratory infection and renal failure, since excretion is via the kidneys.

The striking feature of Umbradil is its water-solubility. Because of this, normal radiograms of the chest can be obtained three to five hours after bronchography (Atwell and Pedersen, 1950). The advantage which this confers needs no comment.

Because of its novel properties the new medium has been given trial on unselected patients at this hospital. Sufficient medium for nine patients was obtained.

Technique

The makers of Umbradil have stated that the medium is intended to supersede iodised oils, and for this reason the technique was identical with that used for iodised oils.

In five patients, 2 ml. of 2 per cent. amethocaine hydrochloride was dropped over the epiglottis after an amethocaine lozenge had been sucked, and 15 ml. of the medium was introduced into the bronchial tree of one lung by the same route.

In the remaining four patients, the crico-thyroid route was used and 0·6 ml. of 5 per cent. cocaine was the local anæsthetic.

As with iodised oils, posturing occupied a period of approximately ninety seconds for each side. Radiological technique was aimed at obtaining a film only slightly harder than a normal chest radiogram. Umbradil *Viscous B* does not cause the dense shadow typical of iodised oils.

RESULTS OF TRIAL OF UMBRADIL

Sex	*Age*	*Comments*	*Technical Result*	*Difficulties*	*Anæsthetic*
M	32	Pt. co-operative	Poor film	Cough++	2% amethocaine hydrochloride via cannula
F	47	Pt. anxious	Examination abandoned	Cough++	
M		Pt. co-operative	Poor film	Cough++ Hæmoptysis Copious expectoration	
M	54	Pt. co-operative	Good	Cough++	
F	18	Pt. very co-operative, and anxious for investigation	Examination abandoned	Cough++ Copious expectoration	
F	47	Pt. co-operative	Good	Cough+	Cocaine 5% via crico-thyroid route
M	45	Pt. co-operative	Examination abandoned	Cough+++	
F	53	Pt. co-operative	Examination abandoned	Cough+++	
F	37	Pt. anxious	Examination abandoned	Cough+++	

Other workers have reported upon the use of Umbradil (see below), but none has mentioned the high rate of failure to obtain a diagnostic film. The opaque medium fails to fulfil the most important property required of it: that of being readily tolerated by the patient.

In all patients coughing was most troublesome, and in five the examination was reluctantly abandoned as impossible. In two further patients coughing was directly responsible for films of little diagnostic value. Only two patients could resist their cough sufficiently to allow good bronchograms to be obtained. Unfortunately, middle lobe filling was incomplete in one, and it was most interesting to note that he and two other male patients complained of no discomfort when immediate re-bronchography with Neohydriol was carried out successfully (Plate 2). This experience was not unexpected, since with iodised oil films of diagnostic value had been obtained in thirty-two consecutive patients in 1951 without any case of total failure arising.

In addition to cough, one patient suffered his first hæmoptysis during the examination and two were troubled by copious tenacious expectoration.

Inspection of the two successful bronchograms (Plates 1 and 3) shows that delineation of the bronchi is very satisfactory. The shadow cast is not, however, very dense and the outline of the smaller bronchi is difficult to study. The rapid absorption of the medium is illustrated by Plate 4.

Contrary to expectations, alveolar filling occurred in two patients. This was probably due to coughing and not directly a fault of the medium.

Discussion

Hellstrom and Holingren (1949) have compared the effects of Umbradil *Viscous B* and its constituents when injected into the bronchi of animals. They found that changes occurred in the alveolar walls similar to those seen when saline was introduced. Unfortunately they did not use iodised oil in their experiments, but the effect would appear to to be similar (Brown, 1928). Clinically they found that Umbradil *Viscous B* caused copious bronchial secretion, thus parallelling the findings in the present investigation. When

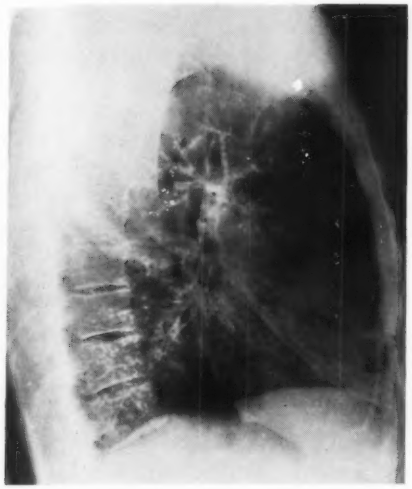

Fig. 1.—Shows Umbradil delineating all but middle lobe bronchi. Definition of smaller bronchi is poor. Alveolar filling is present.

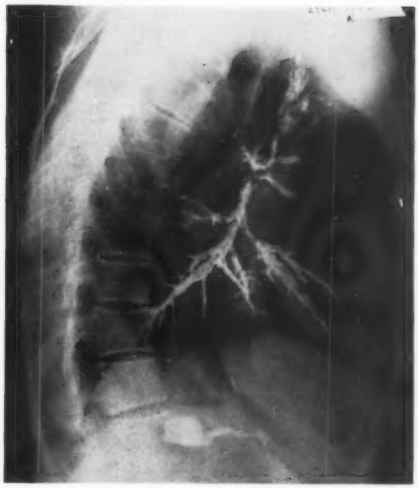

Fig. 2.—Same patient as Fig. 1. Neo-Hydriol bronchogram immediately after the Umbradil bronchogram. The contrast properties of the oily medium are well shown. The alveolar filling caused by the Umbradil has already disappeared.

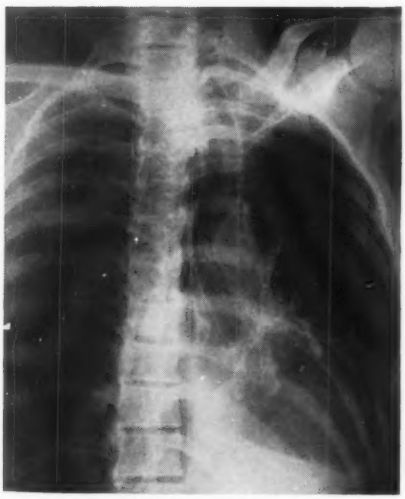

Fig. 3.—Umbradil bronchogram shows evidence of bronchiectasis with stricture and a collapsed left lower lobe.

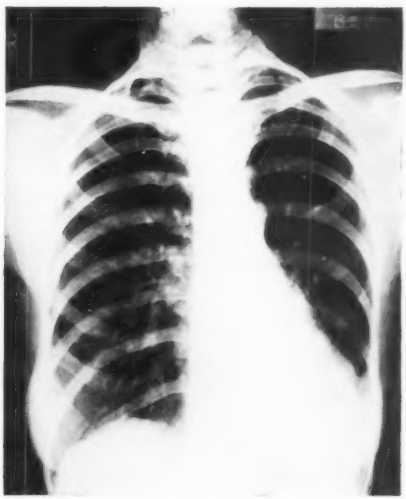

Fig. 4.—Same patient as Fig. 3. Film taken 24 hours after Umbradil bronchogram shows no residual shadows.

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they used unadulterated Umbradil (the radio-opaque principle) ten deaths occurred in a series of fifty animals and dyspnœa and apnœa was observed in some survivors. No deaths occurred when carboxymethyl cellulose or Umbradil *Viscous B* (the trade preparation) were used.

Morales (1949) has stated that Umbradil is unsuitable for myelography, and that it causes painful contractions when injected into the uterus.

In clinical practice many writers have mentioned the difficulty which coughing causes. Two variations in technique have been invoked to reduce coughing. The first method is to use a premedication which many would regard as a surprising prelude to bronchography. Thus Delie and Sourdat (1950) use morphine and atropine, while Atwell and Pedersen use atropine, codeine and nembutal. A second method is to increase the dose of local anæsthetic. Baranger (1950) uses twice the customary dose. Morales (1949) combines both techniques and uses morphine, scopolamine and nembutal premedication and 4-6 ml. of 2 per cent. amethocaine, commenting: " It seems that this procedure is not dangerous " (*sic*).

There are many people in this country who would take exception to the liberal use of local anæsthetic in this fashion. It is agreed that 2 ml. of 2 per cent. amethocaine hydrochloride is the maximum safe dose for bronchographic investigation, and fatalities have followed the use of larger quantities. (When considering the local anæsthetic risk we must bear in mind that the preparation itself contains 0·5 per cent. xylocaine.)

Heavy premedication may be regarded as a more acceptable way of reducing coughing, but evidently some authors do not consider this sufficient by itself. In any case premedication implies the admission of the patient to hospital, and this is a retrograde step at the present time.

The continental investigators use a Matras catheter to produce localised anæsthesia and introduce the medium into specific divisions of the bronchial tree, apparently not attempting to delineate the bronchi of one lung at a single injection.

Do the advantages of Umbradil *Viscous B* merit the adoption of these involved and perhaps dangerous techniques?

It must be recognised that bronchograms are usually made to determine the anatomical localisation of bronchiectasis and to demarcate the diseased from the normal bronchi. To this end it is essential to explore the whole bronchial tree, and it is better for all when this is done at one session. Using iodised oil and given a competent operator, this can satisfactorily be done with little inconvenience. Although there is a possibility of persistent shadows in subsequent radiograms, the risk of post-operative pulmonary collapse is probably no less than with Umbradil, since persistence of its carboxymethyl cellulose vehicle has been demonstrated by Hellstrom and Hollingren (1949).

Conclusion

Although Umbradil *Viscous B* possesses helpful properties not present in iodised oils, its irritant effect upon the bronchial mucosa makes its use difficult. This is stressed by all writers. Baranger (1950) goes so far as to say he will not use the medium in the young or the dyspnœic and that he considers it advisable to have oxygen available when it is used.

It is the opinion of the author that a diagnostic measure should be as free from risk as possible. It is unjustifiable to administer a large dose of local anæsthetic to a patient who may be in no danger of succumbing from his own disease. Yet without liberal local anæsthesia the procedure is one which is difficult for the patient and gives the physician no assurance of success in his investigation.

It is therefore concluded that Umbradil *Viscous B* offers no advantage over iodised oils where exploration of the bronchial tree as a whole is needed. It may, however, be of use where the intention is to explore a specific division of the bronchial tree without the risk of radio-opaque shadows persisting after the investigation. Under these circumstances it is probably advisable to admit the patient to hospital.

Summary

A new radiographic contrast medium for use in bronchography is described. It is more viscous than iodised oil and is water soluble and free from risk of iodism. Its contrast properties are less than those of iodised oil.

In seven out of nine cases its use was unsuccessful because of coughing. Other investigators have experienced similar difficulty.

The danger of administering large doses of local anæsthetic to overcome coughing is stressed.

Where bronchograms are required without the risk of subsequent "lipiodol" shadows it is suggested that the patient be admitted to hospital for suitable premedication before the medium is used.

It is a pleasure to thank the members of the medical and radiological staff of the Royal Northern Hospital. In particular gratitude is expressed to Sister Hathway of the Radiological Department for her technical assistance.

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The supply of Umbradil was obtained from Sweden by courtesy of Duncan and Flockhart, Ltd.

THE TREATMENT OF ARTHROSTEAL TUBERCULOSIS BY RADICAL SURGERY AND THE LOCAL USE OF STREPTOMYCIN

A REPORT ON TWO CASES

By Svante Orell

From the Clinic for Orthopædics and Surgical Tuberculosis at St. Gorans Sjukhus, and the Kronprinsessan Lovisa's Hospital for Sick Children, Stockholm

After the discovery of streptomycin by Waksman in 1944 it was first used in pulmonary tuberculosis by systemic intramuscular injections and later by direct instillation into the cavities of tuberculous empyemata. Its use in arthrosteal infections was a later development. Both systemic and local applications were employed, but only limited success was obtained, since the local lesions were chiefly composed of masses of necrotic tissue with a very poor blood supply. We found that streptomycin showed its best effects only after the removal of these necrotic masses, and that the necessary surgical manœuvres could be completed under cover of streptomycin in combination with other antibiotics and without any dissemination of the tuberculous process (Fig. 1).

The two cases here described illustrate this thesis, and I am satisfied that the results could not have been obtained without surgical removal of the diseased bone.

Case 1. Girl, aged 5. Her illness began with tuberculous pleurisy one year before admission. This appeared to heal with expectant measures, but in June 1949, following whooping cough, she again became unwell and by July manifested mild neck-rigidity. Tuberculous meningitis was confirmed by the finding of acid-fast bacilli in the cerebro-spinal fluid. Following systemic and intrathecal streptomycin she improved steadily and by October was well enough to get out of bed.

She then complained of pain in the right ankle, sufficient to cause her to limp. An abscess slowly developed below the external malleolus and tubercle bacilli were recovered from the pus obtained by incision on December 2. The wound did not heal in spite of rest in plaster, systemic streptomycin sufficient to maintain a blood concentration of 9·8 units per ml., and 5 G. of P.A.S. daily. X-rays (Figs. 2*a* and 2*b*) showed considerable bone destruction and the E.S.R. was 34 mm. at the end of one hour.

On February 8, 1950, the sinus was explored and found to communicate with a cavity in the os calcis as large as a hazel nut. The contained sequestra and granulations were carefully curetted away and the wound was filled with bone chips packed in a mixture of streptomycin (1 G.) and penicillin (200,000 units) in the form of a dry powder. The skin was closed without drainage and plaster immobilisation was continued. Healing was uneventful and consolidation of the graft was complete by November 1950. When seen again in the follow-up clinic in February 1952 the child remained well and the bone focus had completely healed (Fig. 3).

Case 2. Female, aged 28, developed pulmonary tuberculosis in 1948. In September 1949 a skin lesion developed on the left foot and responded to Finsen therapy. In October 1949 there was clinical and radiological evidence of disease in the right hip which, on examination of aspirated fluid, proved to be tuberculous (Fig. 4*a*). She was treated by bed-rest, extension, 10 G. P.A.S. daily, and Conteben 50 mg. twice daily. Between December 21, 1949, and January 20, 1950, the hip was aspirated six times, penicillin and streptomycin being instilled on each occasion.

Daily doses of streptomycin 1 G. and penicillin 500,000 units were given throughout this month, but her general condition deteriorated, an abscess developed round the affected joints and the E.S.R. rose to 50 mm. at the end of one hour. On April 12, 1950, the hip was explored surgically. The abscess was drained, a complete capsulectomy was performed and all carious bone was carefully curetted away from both femur and acetabulum. The joint was filled with a mixture of 3 G. of streptomycin and 300,000 units of penicillin in the form of a dry powder and the wound was closed. Streptomycin 1 G. and penicillin 500,000 units were given intramuscularly for fourteen days. The limb was immobilised in a plaster cast which was changed on June 5 and again on August 2. It was finally discarded in September 1950, five months after the operation, and at the follow-up clinic nearly two years later the joint remained healed and the general health excellent (Fig. 4*b*).

Discussion

Even before the antibiotic era excision of tuberculous lesions in the later stages of the disease was practised with success. The cases described show that, aided by antibiotics, this type of surgery can be successfully undertaken during the stage of invasion. In my opinion the use of streptomycin as a dry powder in the wound is of very great importance, since the massive local dose effect-

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FIG. 1.—DOSAGE SCHEME FOR P.A.S. AND STREPTOMYCIN-PENICILLIN TREATMENT.

Treatment starts and ends with daily doses of P.A.S. of 10 to 14 G. For fourteen days, starting three days before the operation, 1 G. of streptomycin is given daily, intramuscularly, and penicillin (500,000 units in two doses or 300,000 units procain penicillin in one dose). At the time of the operation the dose of P.A.S. must often be reduced or omitted. Penicillin-streptomycin is applied locally (*e.g.*, 1-3 G. streptomycin and 200,000 units penicillin as a dry powder in the evacuated abscess cavity).

FIG. 2.—LATERAL X-RAY PHOTOGRAPHS OF FOOT.

(*a*) Bone lesion the size of a walnut (10-1-50).
(*b*) About one month after operation (6-3-50). Defect in os calcis appears to be smaller than before and is filled with bone implant.

FIG. 3.—X-RAY PHOTOGRAPHS OF FOOT (CASE 1) ABOUT NINE AND A HALF MONTHS AFTER OPERATION (23-11-50).

(*a*) Lateral photograph of os calcis of right foot.
(*b*) Dorsoimplant photograph of os calcis of right foot.
(*c*) Lateral photograph of os calcis, left foot.

Comparison with the previous photographs shows the lesion to be healed.

FIG. 4.—X-RAY PHOTOGRAPHS (22-11-49) OF THE HIP JOINT (CASE 2).

(*a*) Generalised osteoporosis of bone and subluxation of femoral head. Puncture needle inserted.
(*b*) Nine months later.

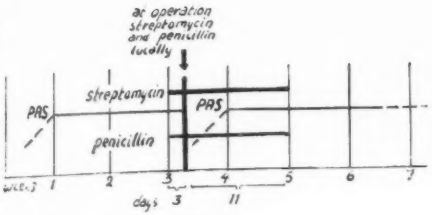

Fig. 1.

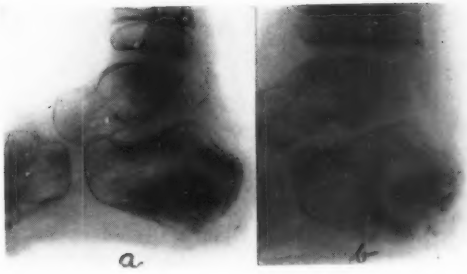

Fig. 2.

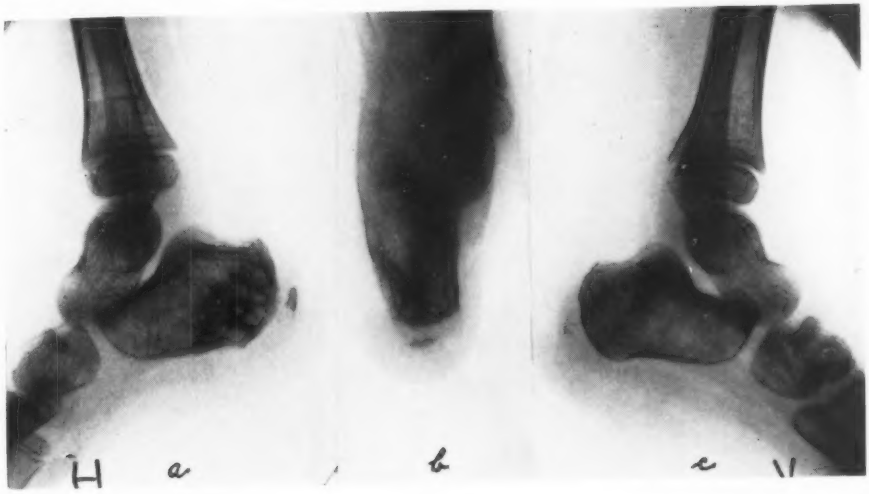

Fig. 3.

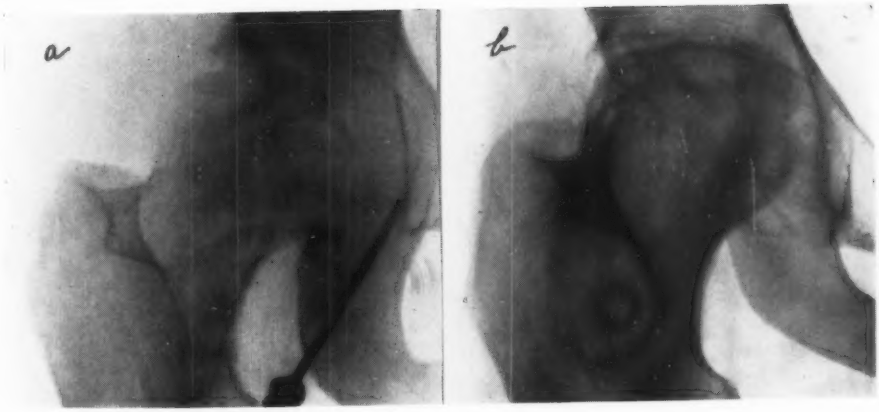

Fig. 4.

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ively controls the activity of any bacilli left in the wound and also prevents any secondary infection from gaining a hold. Moreover, with multiple lesions, general antibiotic treatment is frequently found to secure healing of soft-tissue foci, but is ineffective against arthrosteal disease. This is attributable to the difference in blood supply in the two lesions, and it is not until the bone focus has been excised and fresh granulating tissue exposed to the action of the streptomycin that the full effect of this drug can be obtained. Similar arguments hold good for certain forms of genito-urinary tuberculosis.

Summary

Two cases are described of arthrosteal disease, both associated with considerable local tissue destruction and arising at the same time that a primary focus elsewhere was responding to treatment with systemic streptomycin. In each case general and local treatment with streptomycin and other antibiotics failed to arrest the progress of the metastasis, but after local excision of necrotic tissue and the local application of streptomycin and penicillin in the form of a dry powder, followed by immobilisation, the local lesions healed without complication.

THE INCUBATION PERIOD OF PULMONARY HYDATID CYSTS

By Sidney C. Hawes
From Timaru Hospital, Timaru, New Zealand

Hydatid cyst infestation is a disease that can, and should, be eradicated. It is of major interest in only comparatively few countries, but the many misconceptions concerning the disease warrant publication of the following observations.

Scadding (1950), in his review of Singer's recently published book on the differential diagnosis of chest diseases, questions Singer's statement that hydatid is likely to be mistaken for carcinoma because " of its rapid rate of growth." Scadding thus expresses the general opinion that hydatid cysts are slow-growing, occult tumours; the statement has even been made " a hydatid cyst is as old as its host." Jenkins (1949) has shown that cysts may grow rapidly but, in relation to one of his patients who was only eight years old but had a huge cyst, gave no indication of the " incubation period."

The frequency of routine chest radiography has enabled us to describe three cases in which cyst development, causing symptoms, has occurred within a year.

The first case was that of a man aged 71 who had a prostatectomy in March 1949, before which a chest X-ray (Fig. 1) was taken. In December 1949 he was admitted with pain in the right lower chest and hæmoptysis. His skiagram (Fig. 2) then showed a large abscess which did not alter with postural treatment and penicillin and was found at operation to be an infected hydatid cyst.

The second case had his chest X-rayed in 1949 (Fig. 3) for routine purposes. Nine months later he had another X-ray during investigation of his complaint of effort dyspnœa. Fig. 4, taken four months later and prior to operation, shows the pathognomonic crescents of air between ecto- and pericyst.

A third man, aged 66, was X-rayed in December 1949 after a cardiac infarction. Ten months later persistent cough of some three months' duration led to the development of a cyst at the inner end of the right first rib which might have been expected to cause cough. The patient coughed up a cyst 1 in. in diameter a few days later and has been free of cough since.

The modern techniques of chest surgery may be applied to these cases with great benefit. The cyst is never opened or scraped out or marsupialised (Barrett, 1949). The first case was in hospital for thirty days, which included time taken for treatment of pneumonia surrounding the cyst. The second patient was in hospital for thirty-five days—this time for two thoracotomies. Both men were discharged free of all clinical or radiological evidence of hydatid infestation, and with healed wounds.

All three cases were farmers, and the first two, in spite of their having massive cysts with pericyst rupture, had negative Casoni reactions, though the second case has become positive since his operation.

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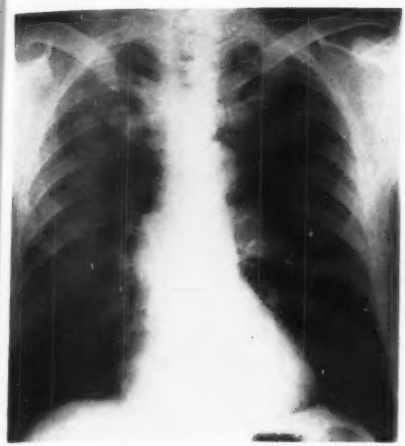

Fig. 1.—Case 1. Some thickening of right clavicle aids identification. X-ray of the chest is within the limits of the normal.

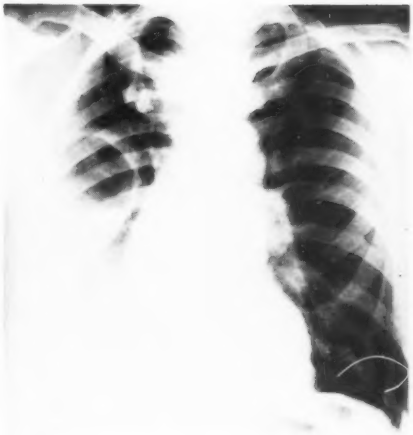

Fig. 2.—Case 1. Appearance eight months later, showing a large abscess due to an infected cyst.

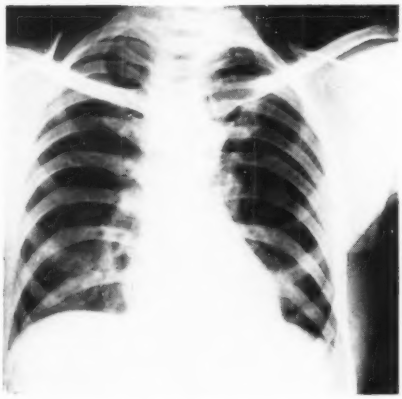

Fig. 3.—Case 2. X-ray of the chest is within the limits of the normal.

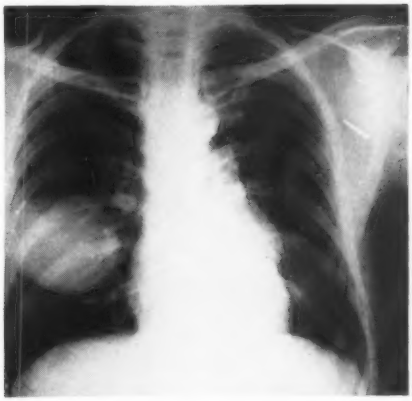

Fig. 4.—Case 2. Showing the crescents of air above two of the cysts.

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REVIEWS OF BOOKS

The Diagnosis and Treatment of Intrathoracic New Growths. By MAURICE DAVIDSON, with a chapter on Radiotherapy by DAVID W. SMITHERS, and a chapter on Operative Treatment by OSWALD S. TUBBS. Oxford University Press. 1951. Pp. 259. 42s.

A superficial acquaintance with the literature of intrathoracic new growths is sufficient to appreciate the complexity of the classification and histological differentiation of these tumours, and the difficulty which attends their satisfactory treatment. The simple cysts alone (and these, with the exception of the dermoid, are mentioned only incidentally) are formidable enough; the solid tumours are highly diverse in structure and the subject becomes, as it were, blurred at the edges when lymphadenoma, with other affections of the reticulo-endothelial system, and adenomatosis of lung are approached. From a first glance at this comparatively slender volume doubt arises as to whether it can cover the ground adequately; but after reading it this doubt no longer remains, for nothing that is of importance seems to have been omitted. It is a very good book.

Conceived from the practical point of view, and with the general reader in mind, it is natural that this monograph should deal more extensively with carcinoma and other malignant tumours than with the benign neoplasms that occur in the chest, although the latter are far from neglected. The differential diagnosis of carcinoma from other tumours, and from other conditions that are not neoplastic at all, is fully considered as part of a campaign for earlier recognition. The importance of early diagnosis is impressively brought out in Mr. Tubbs's chapter, in which the scope of surgical treatment is clearly defined without burdening the reader with too much technical detail. Professor Smithers' contribution on the value of radiotherapy in the management and treatment of this disease will prove revealing to those who are not abreast of the recent developments in this field.

The bare statistical results of treatment of carcinoma of the bronchus may be unpalatable at the present time, but this book serves to stimulate hope for the future, a hope founded upon a synthesis of effort between physician, surgeon and radiotherapist. This synthesis is perhaps not the least of the advantages that have been derived from the recent fruitful collaboration between the Royal Cancer and the Brompton Hospitals.

N. LLOYD RUSBY.

Modern Practice in Tuberculosis. By T. HOLMES SELLORS and J. L. LIVINGSTONE. Butterworth and Co. 1952. Vol. 1, Pp. 354; Vol. 2, Pp. 441. 263 illustrations. 7 gns.

This book is a series of contributions by thirty-nine contributors, the senior editor being responsible for three of the contributions. The editors point out in the preface that it is in no way a comprehensive textbook, but a series of carefully selected sections or chapters, each written by a recognised authority on his or her branch. In this way they forestall and disarm criticism. It is certainly difficult to reach agreement on what should be included in a book of this kind which can be of little use to the initiate and is intended for those with specialised knowledge of tuberculosis. To that end the selections have been good, and although there is a great variation in the quality of the contributions, the standard on the whole is high; that of many is very high,

and some are compact monographs of their subjects. The fusing of isolated contributions into an easy sequence is difficult, and although the editors have given obvious care to their task, there is an air of disjointedness about many parts of the book which might have been partly avoided by an altered grouping of the chapters.

Vol. 1 deals in general with pathological, bacteriological, administrative and preventive problems of pulmonary tuberculosis and contains special chapters on Tuberculosis in Industry, Radiology, Fluorography, Chemotherapy and recent Morbidity and Mortality Statistics. Vol. 2 deals with surgical treatment of tuberculosis of the lungs and contains several excellent and authoritative contributions; in addition there are chapters on Sarcoidosis, Glandular and Abdominal Tuberculosis, Tuberculosis of Bones and Joints, Genital Tuberculosis in the Female, Tuberculosis of the Central Nervous System, Tuberculosis of the Pericardium and Tuberculosis of the Skin, of the Eye and of the Genito-urinary System. Most of these are from well-known men and bear the stamp of experience, judgment and care in presentation. Each chapter has a list of references to the literature and the book should be of great value as a work of reference. It covers a wide field of tuberculosis and places the disease in proper perspective. The volumes are well produced and generously illustrated, but fifteen of twenty-two pages of Chapter 3 were missing from Volume 1 submitted for review. There are a few ordinary mis-spellings, which should be corrected in future editions, and surely the names of Ghon, Birkhaug and Agnes Macgregor are well enough known to make their mis-spelling inexcusable.

C. CAMERON.

Tuberculose Cours de Leysin. Edited by J. MORIN, Privat-docent at the University of Lausanne. 2nd Edition. Paris: Masson et Cie. Pp. 573, with 170 figures, including 5 in colour. 4,000 Fr. fr.

This is a collection of scientific papers written by the phthisiologists of Leysin and Lausanne, and intended to cover most aspects of the field of work in tuberculosis. It appears in the form of forty-two separate articles by twenty-six authors.

The papers are devoted to every aspect of tuberculosis. P. Hauduroy writes on the detection of the tubercle bacillus in the sputum; J.-L. Nicod deals with the pathology of tuberculous disease in the lungs, and makes the point that the early lesion is a benign alveolitis which can resolve completely. Tuberculosis in infancy and childhood are described in detail by M. Jaccottet and J. Morin. The part played by the upper respiratory tract in tuberculosis, a matter often overlooked in this country, is dealt with by A. Barraud, who illustrates his thesis with a macabre tale of the manner in which an over-affectionate wife contracted pulmonary tuberculosis from her infected husband.

The various forms of tracheo-bronchial disease are well described by P. Steiner, and there is an excellent clinical summary of the diagnostic features of the disease by E. Arnold, who stresses the fact that early diagnosis is difficult and that we are still far from perfect in our clinical approach; it is interesting to note that Arnold sums up by stating that " clinical judgment " will always be a most important factor in reaching the correct diagnosis.

J. Morin writes on phrenic paralysis in the treatment of cavities, and considers that there is still a place for this operation in selected cases. He also contributes an excellent commentary on extrapleural pneumothorax.

The medical and surgical aspects of the treatment of all types of tuberculosis

are considered in detail, and it is hardly possible to do more than mention a selection of the contributions here. The volume is well printed on excellent paper and the illustrations are of high standard.

This series of papers is well worthy of study by all who are interested in tuberculosis.

JAMES MAXWELL.

Management of Bronchial Asthma. By H. G. J. HERXHEIMER. 1st Edition. Butterworth and Co. (Publishers), Ltd. 1952. Pp. 107. 22s. 6d.

The author sets out, after a preliminary discussion of the pathology and diagnosis of asthma, to advise the inexperienced practitioner how to find his way " through the undergrowth " of modern therapy in asthma. This is a laudable intention, but he has attempted to say too much in too small a space, and has sometimes lost sight of the audience he has set out to enlighten. Thus, he gives advice concerning the use of adrenalin in a violent attack which might be dangerous in inexperienced hands—namely, to repeat the injection of 20 minims of adrenalin " until relief is obtained " (p. 40). No upper limit for the total quantity to be given in this way is mentioned, and he qualifies his advice by commenting that " more harm is usually done by too small than too great a dosage of adrenalin." Again, after referring to the danger of prescribing morphine in asthma, he says that in asthmatics who " hyperventilate excessively," " depression of the respiratory centre is desirable " and morphine may be the only reliable sedative: this too is dangerous advice, because the interpretation of what is excessive hyperventilation requires a very nice judgment. It is true that all cases of severe asthma are excluded because they are quite incapable of hyperventilating, but in milder cases simple methods are usually effective—*e.g.*, reassurance and suggestion by a confident doctor or the use of barbiturates. Too many deaths have followed the administration of morphine in asthma to encourage its use. Again, several pages are occupied discussing specific desensitisation by exposing the patient to graduated doses of an aerosol preparation of allergen. This is a new treatment still in the experimental stage, requiring complex apparatus, and, as the author explains, a slight misjudgment of the dose may lead to an increase in sensitivity. It is therefore not true to say that it is " one of our few reliable means of treatment " (p. 70). No evidence is produced to show that it is any more reliable than any other treatment, and one could wish that the author had been satisfied by the remark made on a previous page (68), " This method has many limitations and presents great difficulties."

One gets the impression that the author, interested and puzzled by the difficulties in the treatment of asthma, and in the assessment of results which he points out in detail, has allowed himself to think on paper, and has too often confused wishes with fulfilment. One may hope that at a later date this book will be re-written in a more balanced manner. In the meantime, those with an interest in asthma who read this book with adequate discrimination will certainly add to their knowledge.

R. S. BRUCE PEARSON.

Tuberculosis of Bone and Joint. By R. GIRDLESTONE and E. W. SOMERVILLE. 2nd Edition. Geoffrey Cumberlege, Oxford University Press. 1952. Pp. 314, 182 figures. 45s.

All who know Girdlestone's work will regret that he did not live to see the publication of this second edition. The original publication was the

literary product of his life at its zenith in 1940. Unfortunately, at that time most orthopædic surgeons had to think more of war casualties than of tuberculosis of bone, so that the book was read less widely than it deserved and many important adminstrative and clinical facts remained cloistered within the Nuffield Centre or other institutions to which such students were appointed.

It is fortunate that Somerville should have collaborated in the revision at an early stage, although the subject matter is not greatly altered. The book is of great value, and the chapters on general considerations and treatment show the assessment of values after many years of experience. Although alternative methods of treatment are given, details of the authors' own routine are described and enumerated with meticulous care. For instance, six essentials for splintage are described under local treatment of the disease. Again, there is a short chapter on operative treatment in general, but many operations are described under the consideration of individual joints. The reader will be fascinated by the excellent and well-placed illustrations, each one of which is of specific value.

Perhaps the outstanding chapter is that on Pott's paraplegia, which will be of help to the experienced or trainee surgeon, partly on account of the scientific procedure described and also through the extracts from patients' case sheets.

Girdlestone was one of the few of his generation who knew every detail of conservative treatment, but he studied and developed operative treatment for tubercle. Accordingly, there are frequent tables of the indications and contra-indications for such treatment, a valuable contribution to surgery.

The authors are aware that many alternative procedures are available, all of which may lead to the same end result. Some of those chosen appear to be out of date—*e.g.*, Verrall's sacro-iliac graft. Conditions other than tubercle are considered under the heading of diagnosis and furnished with helpful illustrations.

Regarding chemotherapy there is the hesitancy to be expected from a centre which never lags behind but wishes to see end results before advocating a new therapeutic measure.

The Appendix shows the type of plaster and appliances in use and describes how a number of these are made.

This contribution to the literature on tuberculosis is of particular value today and should be familiar to all doing this work. However valuable chemotherapy may prove the surgical treatment will still continue. The authors give credit to Hugh Owen Thomas and Robert Jones and offer proof that the patient with bone or joint tuberculosis can be rehabilitated as a wage earner and a happy member of the community.

ST. J. D. BUXTON.

Essentials in Diseases of the Chest for Students and Practitioners. By PHILIP ELLMAN. Oxford University Press. Pp. xiv+400; 298 illustrations. 30s.

This is a book of convenient handy size at a reasonable price. The paper and print are good, and the illustrations well above the average for textbooks produced in this country since the last war. There are many reproductions of X-ray photographs of the chest, and their value is greatly enhanced by the explanatory line diagrams which accompany them. Good use of line diagrams is also made in illustrating the anatomy of the chest and the varieties of segmental collapse and of encysted effusions.

Dr. Philip Ellman has succeeded in justifying the title of the book as "Essentials in Diseases of the Chest." I do not think there are any important omissions, and it is remarkable how much has been compressed into 400 pages. The various sections are well-balanced and the writing is lucid and easy to read. I doubt if the term "microphotograph" should be used as a caption for a greatly magnified picture of a section: this term was coined to describe photographs of large objects such as the Eiffel Tower, reduced to transparencies the size of a pins' head, and inserted into intriguing little holes in pencils for the delectation of children of the Victorian age. Apart from pedantry, I have no serious criticism of Dr. Ellman's book. It is eminently practical, and bears the stamp of honest experience. It is refreshing to find that the eliciting of the history of a case is given first place in the list of investigations, and that the psychological factor in asthma receives more attention than tests for allergy.

Where there is so much compression there is bound to be provocation by seemingly hasty generalisations—*e.g.*, in the suggestion that early diagnosis and pneumonectomy would so greatly reduce the mortality of bronchial carcinoma.

There is a good index, and no bibliography—how right in a book of this type, and how courageous! Very good value for thirty shillings.

GEOFFREY MARSHALL.

The Quiet Art. A Doctor's Anthology. Compiled by DR. ROBERT COOPE. E. and S. Livingstone, Ltd. 1951. Pp. 283. 12s. 6d.

This volume provides a change from the average book that is sent for review in medical journals. Its very appearance suggests pleasant browsing in a faintly but not academically medical field, and as the reader slowly turns the pages he may find that the few minutes he had proposed to linger may be extended to an hour or more. It is, in fact, a book with restorative powers for the harassed doctor of the mid-twentieth century and one may well ponder whether he will not be the better for an occasional reflective hour.

Dr. Coope is among the medical men whose reading has led him through many ages and many climes, and in this little book he has made it possible for others to follow with infinitely less effort but perhaps with some stimulus for wider reading in the future.

His quotations range from the ancient Talmud to *The Times* of 1948; from Galen, with a neat quip in the eternal battle of physician *versus* surgeon—"chirurgerie is the quick and ready motion of steadfast hands with experience"; to Sir Robert Hutchinson deploring the ultra-scientific outlook which leads men "to the sickroom thinking of themselves as men of science fighting disease, and not as healers helping nature to get a sick man well."

Dr. Coope is not only catholic in his taste and reading, but he has set down his quotations without any obvious plan, so that perusal of this book is a series of surprises. It might perhaps be permissible to criticise the apparent irrelevancy of some of the contents from a purely medical point of view—for example, "when the air comes through a hole, take care of your soul," or Harvey Cushing's portrait of a librarian looking for his umbrella under the letter U in the catalogue might be found in any anthology. It would, however, be churlish to labour this point, since the thread of any anthology is tenuous in the extreme and the purpose of such a volume is reflection rather than pursuit of an object.

The book is divided into eight headings which to some extent facilitate

the search for any particular quotation (although there is an adequate index). Among the vignettes are several charming pen pictures of medical men of the past and present; of Harvey, "As.....mutch, many times governed by his Phantasye," and Sir J. Bland Sutton's personal philosophy, "I divided my life into three parts: in the first I learned my profession, in the second I taught it, in the third I enjoyed it." Particularly pleasing are the dicta referable to sleep, where many words of wisdom are couched in poetic or epigrammatic form. From the Compline is selected "The Lord Almighty grant us a quiet night and a perfect end," while Dickens offers us the following: "There's nothing refreshin' as sleep, sir, as the servant girl said afore she drank the eggcupful of laudanum."

To quote from an anthology is invidious, for each reader must cull his own flowers. Perhaps, however, these random samples will whet the appetite and thus ensure a wide circle of readers for this little book, which every medical man should have at his bedside.

PHILIP ELLMAN.

BOOKS RECEIVED

The following books have been received and reviews of some of them will appear in subsequent issues.

Lung Abscess. By R. C. Brock, M.S., F.R.C.S., F.A.C.S. Blackwell Scientific Publications. Pp. 197. 35s.

Disorders of the Heart and Circulation. Edited by R. L. Levy, M.D. Baillière, Tindall and Cox. Pp. xvi-944, with 368 figures. 91s. 6d.

Colloquium on the Modern Therapy of Tuberculosis. Medical Research Council of Ireland, Trinity College, Dublin.

Laboratory Control of B.C.G. Vaccine, with Special Reference to the Value of some Current Methods of Examination. By E. F. Krohn.

Acta Tuberculosæ Scandinavica, Supplement XXX. Copenhagen: Ejnar Munksgaard. Pp. 181.

An Experiment in the Control of Tuberculosis among Negroes. By Jean Downes. Milbank Memorial Fund, 40 Wall Street, New York 5. 1950. Pp. 67.

Seasonal Differences in Character of the "Common Cold" observed in Two Communities in Westchester County, New York. By Jean Downes. Reprinted from the *Milbank Memorial Fund Quarterly,* January 1952. Milbank Memorial Fund, 40 Wall Street, New York 5.

Incidence of Acute Respiratory Illness among Males and Females at Specific Ages. Study No. 5. By Doris Toucher, Jane E. Coulter and Jean Downes. Reprinted from the *Milbank Memorial Fund Quarterly,* January 1952. Milbank Memorial Fund, 40 Wall Street, New York 5. Pp. 19.

Revista de Sanidad y Asistencia Social, Volume XVI. Ministerio de Sanidad y Asistencia Social, Caracas, Venezuela. Pp. 441.

Tubercle Bacillus and Laboratory Methods in Tuberculosis. By M. A. Soltys in collaboration with C. A. St. Hill and I. Ansell. E. and S. Livingstone Ltd. Pp. 212. 20s.

The Proceedings of the Third Congress on Tuberculosis in Jugoslavia.

REPORTS

Ministry of Health Memorandum (323/Med): Standardisation of Radiological Terminology in Pulmonary Disease and Standardisation of Technique in Chest Radiography. Reports of a joint committee of the Joint Tuberculosis Council, the Faculty of Radiologists and the Society of Thoracic Surgeons.*

We have just received this extremely important report from the Ministry, which we feel should be in the hands of all concerned with the treatment of chest disease. Sir John Charles in a Foreword makes the following observations:

"The maximum benefit from the use of radiography of the chest can be obtained only if those practising it employ the same technique and use uniform terms when interpreting their findings. Accordingly, in March 1950, the Joint Tuberculosis Council, the Faculty of Radiologists and the Society of Thoracic Surgeons appointed a joint committee to consider and report on the standardisation of radiological terminology in pulmonary disease and, later, to bring up to date the report on Standards of Technique in Chest Radiography adopted by the Joint Tuberculosis Council in 1948. These reports should be of great service not only to radiologists but also to all those concerned with the treatment of chest diseases, both in this country and abroad, in securing uniformity in the terms used and in avoiding difficulties and inaccuracies caused by differences in the technique of exposure and processing of films."

Part A of the report, dealing with the Standardisation of Radiological Terminology in Pulmonary Disease, sets out (1) The Scope of the Report, whose object is merely to secure uniformity in the terms used in reporting on radiographs of the chest, not only in this country but also, if possible, internationally in order (*a*) that a report upon a given radiograph may convey a definite meaning to medical practitioners who have not actually seen the radiograph, and (*b*) to give greater precision to terms in common use and to limit the use of synonymous terms. Section (2) of Part A of the Report considers the question of Description or Interpretation; (3), Descriptive Terms, defining mottling, miliary mottling and reticulation; (4), Interpretative Terms, such as infiltration, fibrosis, emphysema, cavity, collapse and atelectasis; (5), Limits of Interpretation, emphasising that it is impossible from a single radiograph to give an opinion as to the degree of activity of disease, hence the importance of serial radiography. Caution is rightly advised in using the word "Normal" in a report, it being emphasised that some such expression as "There is no radiological evidence of disease of the chest" is preferable; (6), Localisation; (7), Nomenclature of the Main Bronchi and Pulmonary Segments. We are pleased to note here that the nomenclature as approved by the Thoracic Society, 1950, is recommended; and (8), Examination and Interpretation of the Radiograph.

Part B of the report deals with the Standardisation of Technique in Chest Radiography. The main factors in securing uniformity are dependent on:

(*a*) The type of equipment, including (1) its power, (2) the kind of tube, (3) the use of a diaphragm and (4) the duration of exposure which is practicable.

* The report is published by Her Majesty's Stationary Office, price 6d.

(*b*) The tube-film distance.
(*c*) The positioning and centring of the patient.
(*d*) The thickness of the patient's chest.
(*e*) The processing of the films.

It is advised that the use of a photo-electric timer would greatly facilitate the attainment of uniformity. Section B of the report considers in detail the Type of Equipment, Variations for Thickness of the Chest and the Processing of Films, and comments upon Intensifying Screens.

From the Report of the County Medical Officer of Health of the London County Council for the year 1950.

Half a Century of Tuberculosis in London

This report of the County Medical Officer of Health contains some interesting observations in relation to " A Review of Changes and Trends since 1900." A retrospective survey, the work of Dr. W. Hartston, Principal Medical Officer (Tuberculosis), reviews the position before 1900, at the turn of the century, and then goes on to consider prevalence of the disease and mortality figures, London's characteristics affecting its tuberculous epidemiology, L.C.C. schemes for the treatment of tuberculosis in 1914, 1922, and 1936, and then studies the effect of two major wars and the National Health Service Act of 1946 and its subsequent evolution.

In a final note on prospects Dr. Hartston makes the following observations:

Tuberculosis in closely aggregated, long-standing, urban industrial communities has become a chronic disabling infectious disease of widespread incidence and heavy though decreasing mortality. The new antibiotic and chemotherapeutic agents show prospects of reducing the infectivity of a proportion of advanced cases and of curing early or localised ones, while B.C.G. and other vaccines offer possibilities of increased immunity for the young susceptible members of the population. The post-war emphasis on " social medicine " has introduced clinicians to a renewed interest in the domestic, financial, psychological and industrial background of the tuberculous patient. Mass radiography and tuberculin surveys are new and useful weapons in epidemiological control. Bovine-type tuberculosis has faded steadily in the last half century both in incidence and severity. Massive scrofulous glands of neck or abdomen, large patches of lupus, wards full of spinal cases fixed to frames, or of children with " hip disease," common at the beginning of the century, are rare conditions now.

The newer pathology envisages the " primary lesion "as a small lung focus in childhood with gross intrathoracic glandular enlargement, usually healing and leaving a partial immunity by allergy which can subsequently break down to produce active phthisis under abnormal nutritional, psychological, traumatic or other injurious tissue influences.

The hazards of exposure to infection once a primary lesion has appeared and healed are, by one current school of thought, no longer considered to have serious implication.

In tuberculosis, as in other diseases, treatment of the individual makes a greater appeal, on the short-term view, than does the application of preventive measures. It may be necessary to emphasise the paramount importance of never losing sight of the fact that tuberculosis is an infectious and preventable disease.

It may well be that the second half of the century calls for a review of the

" set-up " of tuberculosis control and a careful consideration of what further steps can be taken in an attempt to eradicate this great national scourge. This stock-taking may assist in focusing attention on what has been done and what still remains to be done.

BRITISH LEGION VILLAGE—PRESTON HALL

THE report of the British Legion Village at Maidstone for 1950-51 makes interesting reading, and we would like to quote from the observations of the chairman, Lt.-Col. Larking, and of Professor F. R. G. Heaf, its consulting physician.

Col. Larking makes the following statement:

The British Legion Village has now been established twenty-six years. In the medical world the name " Preston Hall " immediately conjures visions of tuberculous ex-Service men recovering from their disability and being employed at useful remunerative work which eventually leads them back to normal life. This generally accepted picture is the result of an intensive effort on the part of the British Legion, combined with the willing help of an enthusiastic staff working with a singleness of purpose for a quarter of a century. Over 10,000 ex-Service men have benefited in the past from the services that have been available at the British Legion Village. It was hoped that the 10,000 would grow in the future to 100,000 or even a million.

But this dream has been rudely shattered by the circumstances that have developed through the application of the National Health Service Act of 1946. This Act separated the responsibility of providing rehabilitation from that of providing treatment, thereby dividing the control of Preston Hall into two parts. The hospital became the property of the Regional Hospital Board and the Industries and the Village were left to the Legion.

This dichotomy would not have been so drastic if it had not meant the divorcement of rehabilitation from treatment in the clinical management of the patients. The result has been that the hospital section is growing steadily into an institution for thoracic surgery and is slowly drifting, as a unit, away from its previous associations with the rehabilitation section. It will not be argued that the provision of a modern chest hospital for the treatment of ex-Service men with tuberculosis is not a necessity, but such a development must not be allowed to influence adversely the growth and progress of the rehabilitation section of the British Legion Village.

The British Legion has played its part and established an efficient rehabilitation unit that provides facilities for training and employment of ex-Service men and women in suitable occupations. It has therefore a right to expect that the authorities responsible for the treatment of the patients will co-operate and encourage the use of these facilities by all the means in their power.

There must be many ex-Service men and women whose treatment cannot be completed without a course of rehabilitation. These are the persons that need the services offered by the British Legion Village. If they could be admitted in the first instance to the hospital for clinical assessment the way for their ultimate return to health and normal life would be made easy. Many of these persons are young and ambitious. It is therefore necessary to concentrate on offering them training courses that will give them opportunities of becoming skilled or semi-skilled technicians, so that they can eventually accept positions in industry that will give them promotion and security in the future.

If the tuberculous are not advised or allowed to take advantage of these

great benefits, then the facilities may have to be offered to those with other disabilities, so far as their clinical condition permits. Rehabilitation is the crying need of the disabled, and the industries at the British Legion Village have been built for the purpose of meeting this need. Maybe the time has come to stop pleading with authorities to use the services that have been provided for the benefit of the patients under their care. It is possible that a new and bold policy is needed that will enable the British Legion to investigate and if necessary expose the reasons why there are over 100 vacant places in the industries awaiting disabled ex-Service men and women; why it is so difficult for ex-patients in all parts of the country to find employment on hospital and sanatorium staffs; and why the broken link between the hospital and the industries cannot be repaired and enable the British Legion Village to function with the same strength as, or even greater strength than it did before July 5, 1948.

Professor Heaf observes:

Man is a creative being. Idleness is both demoralising and depressing and this never more true than during the long days and weeks, maybe months or even years, spent in receiving treatment of chronic diseases such as tuberculosis. If nothing is provided to divert the mind from the disability, it becomes so introspective that the normal healthy desire to create becomes replaced by a definite fear of work that prevents both medical and social progress. It is only necessary to compare patients who have been given suitable occupations with those who have nothing to do to appreciate the value of work as a part of treatment.

At the British Legion Village a scheme has been developed whereby the tuberculous ex-Service man can recover from his disability and at the same time be trained to do useful productive work that will eventually bring him in a good wage and prepare him to take his place alongside his healthy fellows in normal industry when his disease is arrested. Of course it is not a simple matter to build up an organisation that provides these benefits. It takes money, time and skill, and above all the co-operation of the patient himself. But what a fine thing it is to be able to offer such prospects to the disabled! Those fortunate men who have taken advantage of the rehabilitation services at the British Legion Village can face the world again with the knowledge that they are trained in a job that they can do without the constant fear of relapse that is present in the man who goes straight from the sanatorium to the factory. The British Legion Village is the bridge that enables men to cross from the hospital bed to the factory bench and one that leads to the path of promotion and prosperity.

CURRENT STATUS OF ISONICOTINIC ACID HYDRAZIDE IN THE TREATMENT OF TUBERCULOSIS

THE Executive Committee of the American Trudeau Society has reviewed the available evidence on the antituberculous activity of isonicotinic acid hydrazide as presented by Hoffmann-La Roche Inc., E. R. Squibb and Sons, and investigators co-operating with them. On the basis of this evidence, the Committee makes the following statement for the guidance of the medical profession:

1. *Chemical Structure.*—Isonicotinic acid hydrazide is a chemically pure, synthetically produced substance of the general formula $C_6H_7N_3O$. It is

obtained in almost colourless crystals which are highly soluble in water. A closely related derivative, which is also being studied for its antituberculous properties, is the isopropyl derivative. Isonicotinic acid hydrazide is also related to pyrazinamide and to amithiozone.

2. *Activity in Vitro.*—Isonicotinic acid hydrazide is bacteriostatic *in vitro* against *M. tuberculosis* H37Rv in a concentration as low as 0·02 to 0·06 mcgm./ml. It apparently has a very narrow antibacterial spectrum, being ineffective *in vitro* against the common Gram-negative and Gram-positive pathogenic bacteria, against certain protozoa, and against the influenza virus in mice. It may possess some slight antifungal properties.

3. *Activity in Vivo.*—In several species of experimental animals (mice, guinea-pigs, rabbits and monkeys), experiments on the effectiveness of isonicotinic acid hydrazide against tuberculous infection with virulent human strains of *M. tuberculosis* have given promising results in arresting the course of the experimentally produced disease. On the basis of these observations, isonicotinic acid hydrazide appears to be approximately the therapeutic equivalent of streptomycin, at least in the first few months of treatment. Observations on the emergence of strains of tubercle bacilli which may be resistant to isonicotinic acid hydrazide, either *in vitro* or *in vivo*, are meagre, and it is not known if such strains will emerge during treatment or if such emergence will have therapeutic significance. A definite increase in resistance has been obtained *in vitro* with one strain (B.C.G.).

4. *Toxicity and Pharmacology.*—Although the toxicity of isonicotinic acid hydrazide has been determined fairly accurately in several species of animals, some aspects of the pharmacology and toxicology of the drug have not been completely elucidated. On the basis of available studies it appears that both isonicotinic acid hydrazide and its isopropyl derivative are of relatively low toxicity in dosage ranges which appear to be effective. The drugs are apparently largely excreted in the urine. Within an hour or so after administration they appear to be well distributed throughout the body (blood serum, cerebrospinal fluid, pleural fluid).

5. *Dosage.*—On the basis of preliminary studies, the indicated daily dosage is in the range of 3-5 mg./Kg. body weight (150-300 mg. per day for the average adult). This dosage is given by mouth in two or three divided doses. The drug may also be given parenterally.

6. *Toxicity in Man.*—In the dosage range indicated, preliminary observations in man indicate that there is little significant or serious toxicity. The following have been observed, but on a more or less transitory basis, even though drug administration is continued:

(*a*) Constipation.
(*b*) Difficulty in starting micturition (in males especially).
(*c*) Increased reflexes.
(*d*) Positional hypotension and dizziness.
(*e*) Eosinophilia (in about 10 per cent. of cases).
(*f*) Slight drop (0·5-1·0 gm.) in hæmoglobin concentration.
(*g*) Occasional casts and traces of albumen and reducing substances in the urine.

Toxic effects on the eighth cranial nerve, impairment of renal or hepatic functions, or dermatological manifestations associated with the drug, have not been observed so far.

7. *Activity in Man.*—Preliminary observations on the effect of isonicotinic acid hydrazide on the course of tuberculosis in man have been limited largely to patients with far-advanced pulmonary disease, extensive tissue destruction, positive sputum and, as a rule, considerable symptomatology, many of whom have failed to respond or would not be expected to respond to other available therapy. In such patients, treated with 3-5 mg./Kg./day for up to five months of therapy (the majority treated for two to three months), the following changes in clinical course have been observed:

(*a*) Reduction in fever, if present, in two to three weeks, in the majority.
(*b*) Reduction in cough, in the volume of sputum, and in the number of tubercle bacilli raised (as determined by smear). No information is available on conversion of the sputum as determined by culture.
(*c*) Gains in appetite, weight and strength.
(*d*) Some clearing of the reversible component of the pulmonary tuberculous disease by X-ray observation.
(*e*) Initial favourable response has been observed in such non-pulmonary lesions as draining sinuses and fistulæ, mucous membrane tuberculosis, and in a very few cases of miliary and meningeal tuberculosis.

8. *Problems.*—At the present time complete information is lacking on many aspects of the therapy of tuberculosis with isonicotinic acid hydrazide. Among the unknowns are the following:

(*a*) The mechanism of action of the drug on the tubercle bacillus—whether it is tuberculocidal or tuberculostatic; the effect upon the enzyme chemistry of the tubercle bacillus, etc.
(*b*) The mechanism of action upon the host—basically, the precise toxicity in man.
(*c*) The optimal dosage—the number of milligrams per day; whether it needs to be given every day; the optimal mode of administration.
(*d*) The duration of therapy—whether its effect is comparable to that of streptomycin and para-aminosalicylic acid (P.A.S.), indicating relatively long courses of treatment, or whether shorter courses may be as effective.
(*e*) The rate of emergence of drug-resistant strains of tubercle bacilli.
(*f*) The effect of the drug upon the bacteriology of the patient—data are lacking on conversion of sputum by culture; the tissue bacteriology after varying amounts of treatment will need to be studied.
(*g*) The question of potential relapse after initial improvement.
(*h*) The question of whether basic systemic therapy of tuberculosis (especially bed rest) can be modified as a result of treatment with isonicotinic acid hydrazide.

9. *Precautions.*—At present there is no reason to believe that the fundamentals of therapy of tuberculosis should be altered in any way when isonicotinic acid hydrazide is employed. Patients receiving the drug should be hospitalised for careful observation. They should be studied in institutions where potential toxic manifestations may be watched for most carefully and where effects upon the course of the underlying tuberculosis may be carefully observed so that suitable alterations of therapy may be initiated when indicated. Routine laboratory precautions should include frequent blood counts and urinalyses, neurologic examinations, and tests for renal and hepatic insufficiency.

10. *In General.*—The introduction of a new drug in the therapy of tuberculosis is likely to raise more questions for a few years than it will answer. There is no knowledge at the present time that isonicotinic acid hydrazide or its

isopropyl derivative will accomplish more than has been accomplished with streptomycin and P.A.S. It may prove to be an additional drug of great value. It may be years before its exact contribution to the therapy of tuberculosis can be assessed accurately. A large reservoir of undetected and untreated cases of active tuberculosis exists throughout the United States, and there is every expectation that, in spite of the more effective chemotherapy of tuberculosis currently available, the need for hospitalisation in institutions with qualified personnel and adequate laboratory facilities will increase rather than decrease. There is at present no basis for expecting that isonicotinic acid hydrazide, or any other drug available, can safely be counted upon to reduce the duration of hospitalisation. Rather, in most instances, at least, it may lead to prolongation of hospital treatment, since effective chemotherapy may facilitate desirable forms of therapy not otherwise possible.

It should be emphasised strongly that, with more numerous effective antituberculous compounds available in the treatment of tuberculosis, more intensive case finding than ever will be indicated. Only through this means can maximum advantage be taken of improvements in therapy.

11. *Summary.*—After a review of available data on the action of isonicotinic acid hydrazide and its isopropyl derivative upon the tubercle bacillus *in vitro*, and upon the course of experimental tuberculosis in animals and clinical tuberculosis in man, it may be stated that their demonstrated action, although highly encouraging, appears in no way to alter the basic principles of the treatment of tuberculosis as presently understood. Much more work will need to be done to ascertain the exact place of these drugs in the treatment of the disease. With several carefully co-ordinated studies in prospect, it is anticipated that further information will accumulate rapidly.

CONGRESS ON TUBERCULOSIS IN JUGOSLAVIA

We have received the Proceedings of the Third Congress on Tuberculosis in Jugoslavia, which took place in the autumn of 1950. Among the subjects which were discussed were those of Hilar Tuberculosis in Adults, by Professor Spiro Janovic; Hilar Tuberculosis in Children, by Dr. Ljubomir Vulovic; the Results of Treatment of Pulmonary Tuberculosis, by Dr. Stanko Ibler; the Critical Approach to New Drugs in Tuberculosis, by Dr. Marc Daniels; the Results in Operative Treatment of Joint and Bone Tuberculosis, by Dr. Alexander Manzoni; Health Education at the Anti-tuberculosis Dispensary, by Dr. Gligor Muratovski; Statistical Analysis of the Work of the Anti-tuberculosis Dispensaries, by Dr. Bojan Pirc; and Protection of the Staff in Anti-tuberculosis Establishments, by Dr. Fran Janjic.

NOTES AND NOTICES

ROYAL SANITARY INSTITUTE HEALTH CONGRESS TO MEET IN HASTINGS IN 1953

THE Royal Sanitary Institute has accepted an invitation from the Corporation of Hastings to hold the 1953 Health Congress in Hastings from Tuesday, April 28, to Friday, May 1, inclusive. Forty nations were represented at the 1952 Congress, which was attended by 2,140 experts on health. Amongst them were five delegates from the Soviet Union specially charged with the mission of investigating British public health methods.

MINISTRY OF HEALTH

TUBERCULOSIS

WE have received the following communication from the Ministry dated April 15, 1952:

1. The Minister is informed that in some areas there is evidence of a lack of co-operation between chest clinics and Local Health Authorities which is impeding the authorities in carrying out their responsibilities under Section 28 of the National Health Service Act for the prevention of tuberculosis and for the care and after-care of tuberculous persons. The Minister is confident that the Boards share the view which he has previously urged [see RHB(48)9] that the closest co-operation between all concerned in the tuberculosis service is essential to the proper management of the individual case and to the control and prevention of the disease. He would therefore request Boards to review the present position in their respective Regions, and to take steps, where necessary, to ensure that the work of chest clinics on the diagnostic and treatment side is everywhere fully integrated with that of Local Health Authorities as regards prevention and after-care. They may consider it desirable to conduct this review jointly with each Local Authority in the Region.

2. It appears that, in particular, difficulty has been experienced by some Medical Officers of Health of Local Health Authorities in obtaining from Chest Physicians in charge of clinics the information required for the authority's purposes contained in chest clinic registers or other case records. Clearly no such difficulty should be allowed to continue; and it should be impressed on all physicians in charge of chest clinics that it is part of their duty to give the Medical Officer of the Local Health Authority any information he may reasonably require, or to allow him ready access to it at the clinic, so that he may be able to carry out his duties as freely and fully as when the clinic services were formerly conducted by a whole-time member of his staff.

3. It has also been represented to the Minister that not all chest physicians are sufficiently concerning themselves in the preventive and after-care aspects of tuberculosis work, and that there is a tendency to give less regard to this than to clinical work. The Minister would remind the Boards of what was said on this point in paragraph 7 of RHB(50)64 and would ask them to do everything they can towards counteracting this tendency; and to emphasise the vital importance of the part that should be played by the chest physician, in association with the Medical Officer of Health of the Local Health Authority,

in the preventive and after-care arrangements for which that authority is responsible.

4. The Minister would also take this opportunity of emphasising the requirement contained in paragraph 1 of RHB(50)22 that information should be sent to the Medical Officer of Health of the Borough or Urban or Rural District concerned on the admission to, or discharge from, a sanatorium or hospital of a patient suffering from a notifiable disease, *including tuberculosis*: and he would urge the Boards to ensure careful observance of this requirement.

N.A.P.T.

CLINICAL CONFERENCE IN SCOTLAND

OCTOBER 3 AND 4, 1952, IN THE ART GALLERIES, PERTH

Open to all doctors interested

PROVISIONAL PROGRAMME

Friday, October 3

9.30 a.m. "The Choice of Surgical Methods in the Treatment of Pulmonary Tuberculosis and Allied Conditions."

Speakers: Mr. Bruce Dick (Consultant in Thoracic Surgery, Western Regional Hospital Board).
Dr. N. Lloyd Rusby (Physician to the London Hospital, and the London Chest Hospital).
Mr. Vernon Thompson (Surgeon to the Department of Thoracic Surgery, London Hospital, and to the London Chest Hospital).
Mr. Andrew Logan (Consultant in Thoracic Surgery, South-eastern Regional Hospital Board).

2.30 p.m. "Antibiotics and Other Recent Methods in the Treatment of Pulmonary Tuberculosis."

Chairman: Professor John Crofton (Edinburgh University).
Speaker: Dr. Andrew Morland (Physician, University College Hospital, London).
Dr. D. G. McIntosh (Senior Tuberculosis Physician, Eastern Regional Hospital Board) will open the general discussion.

Saturday, October 4

9.30 a.m. "The Protection of Nurses and Medical Students against Tuberculosis."

Speakers: Dr. F. A. H. Simmonds (Medical Superintendent, Clare Hall Hospital, Middlesex).
Dr. Tobias Gedde-Dahl (Secretary, Norwegian Association for the Prevention of Tuberculosis).
Miss I. M. Gourlay (Matron, Mearnskirk Hospital).

2.30 p.m. Meeting of Sanatorium Matrons and Nurse Teachers to be addressed by Dr. Tobias Gedde-Dahl.

Fee: The fee for the course is two guineas, and this should be paid to Miss A. J. Weir, N.A.P.T., 65, Castle Street, Edinburgh 2 (Central 6527).

AMERICAN COLLEGE OF CHEST PHYSICIANS

THE Twelfth Congress of the International Union Against Tuberculosis and the Second International Congress on Diseases of the Chest sponsored by the Council on International Affairs, American College of Chest Physicians, will be held in Rio de Janeiro, Brazil, August 24-30, 1952. Both Congresses will be held under the Presidency of Professor Manoel de Abreau; Dr. Reginaldo Fernandes will serve as general secretary. The Honourable Getulio Vargas, President of Brazil, has accepted the honorary chairmanship of the Congresses.

MEDICAL TREATMENT ABROAD

THE Secretary to the Exchange Control Medical Advisory Committee advises the Editor that, owing to the increased shortage of foreign currency, the Treasury is compelled to ask the Committee to reconsider the policy under which currency certificates are granted to patients seeking treatment abroad.

Until recently currency was made available in countries of the European Payments Union in all cases of real illness, whether or not comparable treatment could be obtained at home or within the sterling area. In future, however, the normal rule will be to allow currency certificates only where the required treatment cannot be had in this country or elsewhere in the sterling area. Comparative lengths of journey will be taken into account. Patients suffering from tuberculosis will usually be allowed currency and, during the winter months only, short visits for convalescence after serious illness may be permitted, especially in patients over 65. Currency will still be allowed for a husband or wife to accompany a sick partner abroad if either of them is over 60, and in certain cases for companions to patients.

The procedure is simple. The patient's doctor sends the application, with particulars of the case, direct to the Secretary, Exchange Control Medical Advisory Committee, Tavistock House North, Tavistock Square, London, W.C.1.

The Committee continues to be grateful to the medical profession for their support in this difficult task, and although the number of applications granted is likely to be smaller, they are satisfied that it will still be possible for the seriously ill to obtain any essential foreign treatment.